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SUBTOTAL GASTRECTOMY

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This paper is concerned with the immediate postoperative appearance of the remnant of the stomach following subtotal gastrectomy. Twenty-one cases are presented. For each patient a fluoroscopic examination was made by Dr. L. W. Paul, of the department of radiology, to check the mechanics of evacuation of the barium sulfate from the remnant of the stomach. The roentgenogram taken at the completion of the fluoroscopic examination in each case is reproduced here as a black and white copy of the original film. All gastrectomies were performed in an identical manner. Evaluation of the operation from the standpoint of the clinical course of the patient as well as the mechanics of stomach evacuation is thus possible. Technical detail has purposely been omitted in favor of emphasizing important points of the operation.

METHOD

Figure 1 presents diagrammatically the method of performing the operation. In A is shown the method of placing the clamps; the angle for placing the second clamp should be especially noted. B shows the inversion of the superior portion of the stomach along the lesser curvature.

Figure 2 is a diagrammatic representation of the completed operation. The antecolic loop of jejunum is actually much shorter than depicted. Measurement of this loop from the ligament of Treitz to the beginning of the anastomosis has been ascertained as between 9 and 14 inches (23 and 36 cm.). In addition, the proximal jejunal loop will be decreased in length after the anastomosis has been completed. This is due to the increased tonus as a result of stimulation. A 14 inch loop has been found to decrease 2½ inches (6.35 cm.) in length. The proximal (afferent) loop is brought to the lesser curvature in all cases. The gastric stoma is placed on the side of the greater curvature. The emptying of the stomach into the distal (efferent) loop is facilitated by these two steps. Suturing the proximal loop along the lesser curvature will buffer the dangerous angle and prevent (to some extent) dumping into the proximal loop. The filling of the distal loop is illustrated by the black and white reproductions. Extreme care in the closure of the duodenal stump cannot be overemphasized. A routine method is not followed. If a good length of duodenum can be obtained, a Clute clamp is used. A small Payr clamp with a Parker-Kerr stitch serves equally well. If the duodenal stump is short, an "open" type of inversion is performed, a Connell stitch being used. There is experimental evidence to prove that the latter type of duodenal closure is fol-

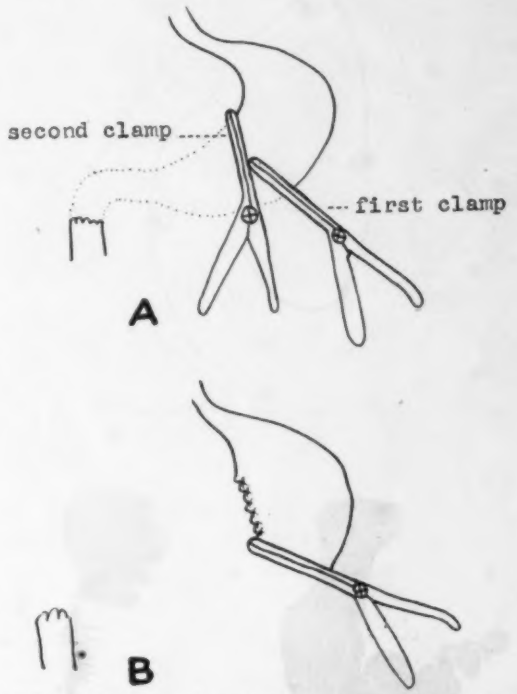

Fig. 1.—Method of performing the operation. *A*, position of clamps on the proximal portion of the stomach (note the angle for placing the second clamp); *B*, inversion of the superior portion of the stomach along the lesser curvature.

lowed by the fewest number of duodenal "blow-outs." If the safe removal of a duodenal ulcer is impossible, a Lahey gastroenterostomy [1] or a Finsterer exclusion resection (with antral excision) is performed. Removal of the ulcer

1. Lahey, F.: Simple, Useful Anterior Gastro-Enterostomy, Surg., Gynec. & Obst. **78**:169 (Feb.) 1944.

later by a second operation, as advocated by both Lahey [2] and McKittrick,[3] should be given serious consideration.

RESULTS

Figures 3 to 8 are black and white reproductions of the postoperative remnant of stomach and the adjacent small bowel. There are 4 cases of gastric ulcer, 5 cases of duodenal ulcer, 11 cases of carcinoma and 1 case of localized Hodgkin's disease.

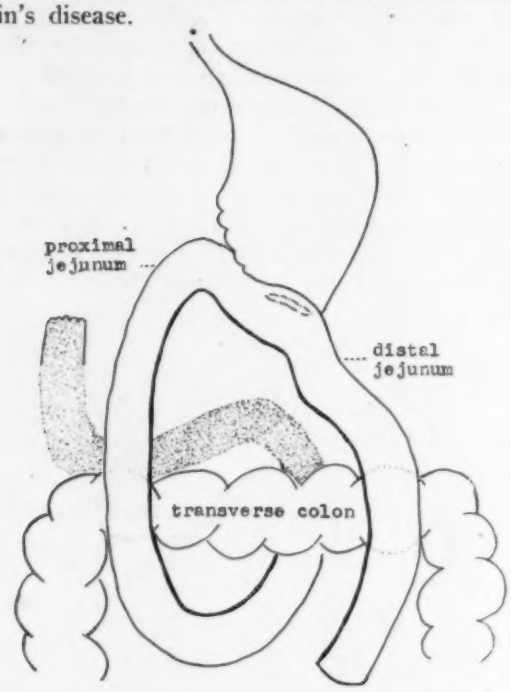

Fig. 2.—Diagrammatic representation of the completed operation.

Fig. 3.—*A*, stomach before operation; a duodenal ulcer deformity is indicated at *d*. *B*, stomach after operation; the proximal loop of jejunum is indicated at *a* and the distal loop at *b*.

Figure 3 consists of photographs of the india ink drawings taken directly from the original roentgenograms. They illustrate the appearance of the stomach before and after resection for duodenal ulcer. To be noted are the amount of stomach resected and the failure of visualization of the proximal jejunal loop. The fluoroscopic examination was reported as indicating immediate emptying through the stoma, with rapid evacuation of the remnant of stomach.

In the remaining illustrations the cases of peptic ulcer have been grouped first and the cases of carcinoma second. The preoperative roentgenograms have been omitted. The postoperative roentgenograms are accompanied by the report of the fluoroscopic examination.

Fig. 4.—Postoperative appearance of the gastric remnant following resection for peptic ulcer (*A*, *C* and *D* for gastric ulcer and *B* for duodenal ulcer). The proximal jejunal loop is indicated at *a* and the distal loop at *b*.

The postoperative appearance of the remnant of stomach following resection for peptic ulcer is shown in figure 4. *A*, *C* and *D* are from cases of gastric ulcer and *B* from a case of duodenal ulcer. In the cases represented in *A*, *B* and *D* the fluoroscopic examination revealed immediate emptying through the stoma, with rapid evacuation of the remnant of stomach; in the case

2. Lahey, F.: The Use of an Identifying "T" Tube in the Common Bile Duct in Gastric Resection for Duodenal Ulcer Adherent to the Bile Ducts, Surg., Gynec. & Obst. **80**:197 (Feb.) 1945.

3. McKittrick, L. S.; Moore, F. D., and Warren, R.: Complication and Mortality in Subtotal Gastrectomy for Duodenal Ulcer: Report on a Two Stage Procedure, Ann. Surg. **120**:531 (Oct.) 1944.

represented in *B* there was slow emptying through the stoma, but evacuation was satisfactory. This group is interesting from the standpoint of prediction of the efficacy of the anastomosis as related to the appearance of the stomach remnant. The ballooning of the remnant as shown in *B* is usually an indication that the remnant empties slowly. The failure of the remnant to fill completely (as in *C*) plus a heavy outline of the distal loop usually indicates rapid emptying of the stomach. Paradoxically, the opposite was true in these 2 cases, as will be noted from the report of the fluoroscopic examination.

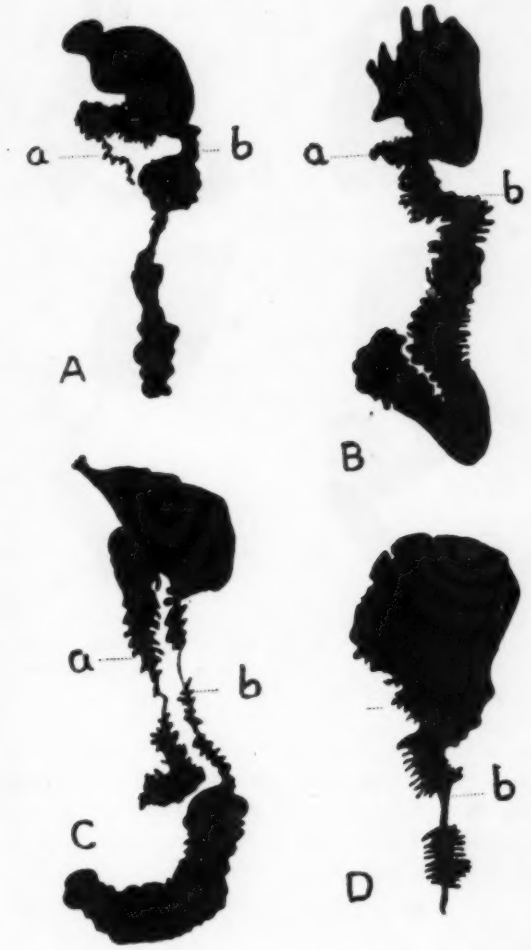

Fig. 5.—Postoperative appearance of the stomach after resection for ulcer (*A*, *C* and *D* duodenal and *B* gastric). The proximal jejunal loop is indicated at *a* and the distal at *b*; in *D* the proximal loop was not outlined.

Figure 5 *A*, *C* and *D* show the postoperative appearance of the stomach after subtotal gastrectomy for duodenal ulcer, and *B*, for gastric ulcer. In all these cases the fluoroscopic examination was reported to show immediate emptying through the stoma with rapid evacuation of the stomach remnant.

Figure 6 shows results in 4 cases of carcinoma: In the first (*A*) fluoroscopic examination revealed immediate emptying through the stoma but slow evacuation of the remnant of stomach. In the second (*B*), emptying did not occur immediately,

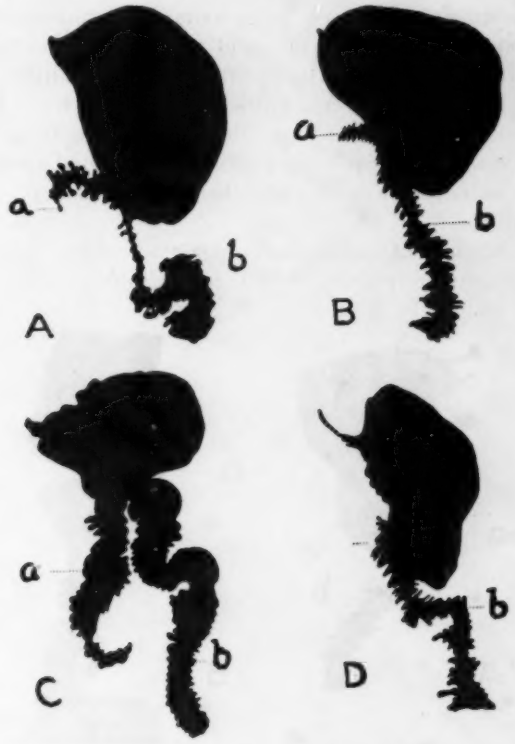

Fig. 6.—Postoperative appearance of the stomach in 4 cases of carcinoma. The proximal jejunal loop is indicated at *a* and the distal at *b*. In *D* the proximal loop was not outlined.

and there was 50 per cent retention at the end of five hours. In the third case (*C*) there was slow emptying through the stoma, with satisfactory evacuation of the stomach remnant, and in the fourth, immediate emptying through the stoma, with rapid evacuation of the stomach.

Figure 7 presents results in 3 cases of carcinoma (*A*, *C* and *D*) and 1 case of Hodgkin's disease (*B*). In all these cases immediate emptying through the stoma with rapid evacuation through the remnant of stomach was revealed.

In figure 8 are shown the results in 4 cases of carcinoma, in all of which fluoroscopic examination revealed immediate emptying through the stoma with rapid evacuation of the remnant of stomach.

In reviewing the foregoing statements it will be noted that in 4 cases fluoroscopic examination was reported to reveal slow immediate emptying of the remnant of stomach. Thus in

only 80 per cent (17 of 21) was the anastomosis considered to be "ideal." The term "ideal" denotes an anastomosis in which immediate emptying of the remnant of stomach is apparent and is followed thereafter by rapid evacuation. In only 1 of these 4 cases were both immediate emptying and eventual evacuation of the gastric remnant entirely unsatisfactory. Despite this, the patients' subjective symptoms were minimal, consisting mainly of mild epigastric distress which eventually disappeared. In the other 3 cases results were clinically satisfactory. If these 3 cases may be added to the original 17 cases of "ideal" operation then satisfactory anastomosis was accomplished in 95 per cent of the cases.

Studies of postoperative gastric acidity were unfortunately carried out in only 6 of the 9

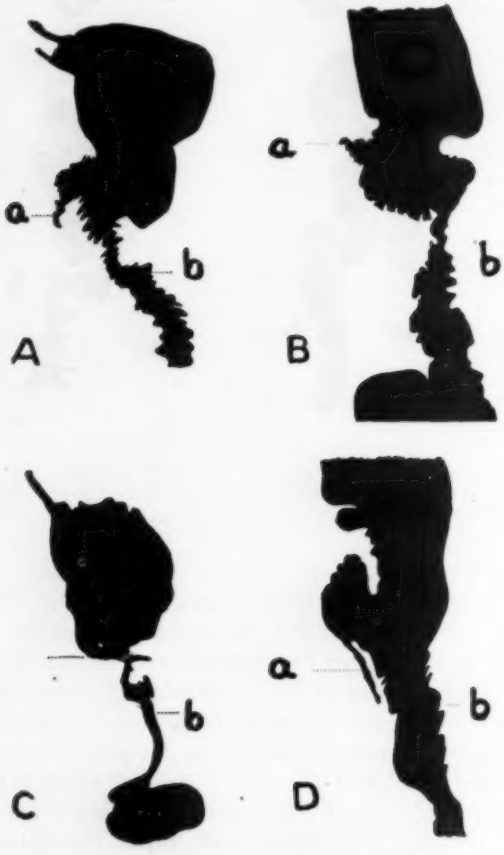

Fig. 7.—Postoperative appearance of the stomach in 3 cases of carcinoma (*A*, *C* and *D*) and 1 case of Hodgkin's disease (*B*). The proximal jejunal loop is indicated at *a* and the distal at *b*. In *C* the proximal loop was not outlined.

cases of peptic ulcer. Nevertheless, it is worthy of note that free hydrochloric acid was not found on any of the postoperative determinations. Furthermore, recurrent ulceration has not been a complication, despite the fact that the anastomosis was performed some distance from the ligament of Treitz. Additional resections, reports of which are not included in this study, have also been notable for the extreme rarity of recurrent ulceration.

The time required for performance of the operation is of more than passing interest. It

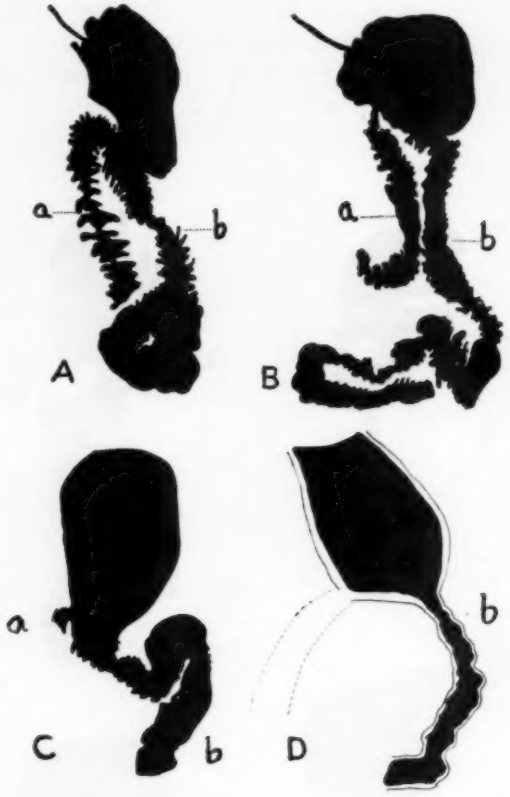

Fig. 8.—Postoperative appearance of the stomach in 4 cases of carcinoma. The proximal jejunal loop is indicated at *a* and the distal at *b*. In *D* the proximal loop was not outlined.

has been said that some surgeons require six hours. Hans von Haberer and De Petz [4] found forty-five minutes to be ample time. Two and one-half hours was the average time per case in the gastrectomies herein reported.

COMMENT

In examining the postoperative black and white reproductions and evaluating them with the clinical course of the patient an interesting fact becomes apparent. Those patients exhibiting rapid evacuation of the barium from the remnant of stomach have had the most satisfactory postoperative course. This leads to the belief that an anastomosis resulting in a "dumping" stomach

4. de Petz, A.: Aseptic Technic of Stomach Resections, Ann. Surg. **86**:388 (Sept.) 1927.

is the ideal operation to perform. This statement must be qualified to some extent. Not only must the anastomosis result in a dumping stomach, but the evacuation must be predominantly, if not totally, into the distal (efferent) loop. If the process is reversed, then the increased enteric pressure gradients resulting may secondarily prevent satisfactory evacuation of the gastric remnant.

The presentation of these cases of antecolic anastomosis should not be construed as an attempt to recommend this type of operation to the exclusion of any other type. We frequently use short loop retrocolic anastomoses with excellent results. Wangensteen [5] favors the latter operation to the exclusion of all others. His experimental observations and clinical results give testimony as to the efficacy of the method. Nevertheless, surgeons will continue to disagree as to which method is the most satisfactory one. The failure to agree on a standardized technic indicates that each method must have its objectionable features. Individual preference will always militate against standardization of the operation. The emphasis should be placed on the performance of the best type of operation in keeping with the greatest margin of safety to the individual patient. One "routine" procedure followed by death of the patient will not be rectified by any number of subsequent perfect operations. Each case presents its own problems, and the following is an illustrative one. A subtotal gastrectomy had been planned for a patient suffering with duodenal ulcer. This was altered to a Lahey gastroenterostomy when the patient exhibited a generalized convulsion at the beginning of the operation. The postoperative course of the patient was uneventful. There was complete relief of symptoms plus a gain of 40 pounds (18 Kg.) in weight. One is justifiably skeptical that a subtotal gastrectomy would have done so well. An opinion once expressed by Dr. Donald Balfour [6] on this point will bear repetition: "The soundest argument for so called conservative surgical treatment in peptic ulcer is that such methods when intelligently applied yield very satisfactory results and these conservative measures will probably remain an important part of the surgical treatment of the disease as long as surgery of peptic ulcer is required." Briefly, then, the decision as to method should depend on the nature of the primary disease, the amount of stomach to be removed, the surgeon's skill and divers other factors that vary with the patient's age, diet and mental attitude.

The late results of subtotal gastrectomy should be the concern of every one. Ingelfinger [7] has recently summarized the literature, and the following are his conclusions: Thirty per cent of patients do not regain their normal weight, and some do not regain their preoperative weight. Ten per cent suffer from symptoms soon after taking a meal, particularly if the meal is large. These symptoms consist in epigastric distress, pressure, belching, nausea and weakness. Some patients have soft stools, but few suffer from persistent diarrhea. Carbohydrates and proteins are usually adequately absorbed but some impairment in absorption of fats may occur. The dextrose tolerance test shows a rapid rise in blood sugar followed by a fall that may reach hypoglycemic levels. In a few cases symptoms occur during the hypoglycemic phase. Pernicious anemia is an extremely rare sequela, but normochromic or hypochromic anemia is not infrequent. The gastric remnant is at first small, gradually enlarges and assumes some of the reservoir function of the stomach. If the gastric remnant is extremely small, the small bowel dilates to form reservoirs near the anastomosis. Even if the stoma is anatomically perfect, gastric evacuation after subtotal gastrectomy may be delayed by functional disorders. A nourishing diet, taken frequently in small quantities fortified with vitamins and iron, often alleviates some of the complications of gastrectomy. Symptoms and various motility disorders which may be prominent during the first postoperative year subsequently tend to correct themselves spontaneously.

The only exception to these conclusions has to do with the dilatation of the remnant of stomach. We have good reason to believe that the remnant will decrease in all cases of subtotal gastrectomy if the anastomosis is functioning in an "ideal" manner. One patient in this series has now been followed over a period of thirty-one months. Series of roentgenograms of the upper part of the gastrointestinal tract have been recorded every six months. These studies reveal a gastric remnant decreased in size. The barium empties immediately through the stoma, with no evidence of any reservoir function of the remnant. Only if there is a slow emptying will dilatation result. Increased enteric pressure gradients supposedly play some part in slow emptying of the stomach. It seems logical to assume that slow emptying, whether on a basis of partial obstruction at the

5. Wangensteen, O. H.: The Criteria of a Satisfactory Operation for Ulcer and Causes of Failure After Gastric Resection for Ulcer, Minnesota Med. **28**:66 (Jan.) 1945.

6. Balfour, D. C.: Surgical Treatment of Peptic Ulcer, Surgery **2**:732 (Nov.) 1937.

7. Ingelfinger, F. J.: The Late Effects of Total and Subtotal Gastrectomy, New England J. Med. **231**: 321 (Aug. 31) 1944.

outlet or increased intestinal pressure gradients, has a common root in the method of anastomosis. Dilatation could thereby be an indication of faulty anastomosis.

Occasionally a statement is made with apparent conviction that subtotal gastrectomy often ruins a patient for life. If such were actually true, little could be said for those surgeons persisting in use of such a procedure. To the infrequent person who is pessimistic regarding the outcome of the operation the following quotation [7] may be of some comfort: "Even when the undesirable sequelae of gastrectomies are marked, they rarely interfere with the patient's ability to work, and practically never are they so dangerous or distressing as the disease for which the gastric resection was undertaken."

CONCLUSION

Twenty-one cases of subtotal gastrectomy utilizing an antecolic anastomosis were studied by means of postoperative roentgenologic examinations with barium sulfate. These studies indicate that the best anastomosis is one which effects a rapid emptying through the gastric stoma with evacuation of the stomach predominantly into the efferent loop. If this is accomplished, it is believed the stomach remnant will always decrease in size during the postoperative period.

Our results offer corroborative evidence to substantiate the established fact that the "long loop" antecolic method of anastomosis is a satisfactory one.

POSTOPERATIVE GOUTY ARTHRITIS

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AND
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Modern surgery has advanced beyond the confines of the operating theater. Surgical knowledge is progressing so rapidly that the surgeon must of necessity be mindful of the results of laboratory investigations. In recent years the role of chemistry has become more closely associated with surgery. Through chemical analysis many postoperative complications have been explained.

Innumerable authors have stressed the importance of protein to the surgical patient. The excreted daily. This (multiplied by 6.25) means that 125 to 250 Gm. of protein is lost per day.[1] Thus it seems that endogenous protein metabolism in surgical patients is proceeding more rapidly at rest than during normal activity. One of the products of this endogenous protein metabolism is uric acid.

When uric acid is the end product of body purine metabolism, it is called endogenous uric acid. The term endogenous is used to distinguish

Salient Features in Four Instances of Postoperative Gouty Arthritis

Case	Age	Sex	Diagnosis	Operation Performed	Post-operative Appearance of Gout	Joint Involved	Osteoarthritis Present	Blood Uric Acid, Mg. per 100 Cc.	Plasma Protein, Mg. per 100 Cc.	Residual Joint Manifestations
Recorded at Kings County Hospital, Brooklyn										
1	56	M	Gastric carcinoma	Subtotal gastrectomy	Fifth day	Left large toe	Slight	5	5	None
2	61	M	Rectal carcinoma	Abdomino-perineal resection	Third day	Right large toe	Moderate	5.2	4.5	Superficial desquamation of skin
3	57	M	Gastric carcinoma	Subtotal gastrectomy	Fourth day	Right large toe	Moderate	5.5	4.5	None
Recorded at Lahey Clinic (May 1937)										
4	60	M	Benign prostatic hypertrophy	Transurethral prostatic resection; bilateral vasectomy	Fourth day	Right thumb	Severe	5.4	?	Soft tissue swelling

removal of protein from the diet almost immediately produces an alteration in plasma protein. Well nourished surgical patients who are deprived of an adequate protein diet can easily approach a state of acute starvation during hospitalization.[1] In these circumstances the patient must replenish his protein deficiency through endogenous protein secured from his own body. This loss of protein tissue can be measured in the terms of nitrogen excretion. Studies have revealed that after operation as much as 20 to 40 Gm. of nitrogen may be

the uric acid formed from the purines produced in the course of the digestion of food proteins. Endogenous uric acid is derived from the breakdown of nuclear material found in muscle tissue and glandular structures.[2] An increase in endogenous uric acid produces an increase in blood uric acid above the normal level (3 mg. per hundred cubic centimeters of serum or whole blood). This hyperuricemia is similar to that seen in gout. These facts seem pertinent in view of our personal observation of 4 patients

From the Department of Surgery, Lahey Clinic.

1. Elman, R.: Acute Starvation Following Operation or Injury, with Special Reference to Caloric and Protein Needs, Ann. Surg. **120**:350 (Sept.) 1944.

2. Best, C. H., and Taylor, N. B.: Physiological Basis of Medical Practice, Baltimore, Williams & Wilkins Company, 1943. Jones, W.: Nucleic Acids; Their Chemical Properties and Physiological Conduct, London, Longmans, Green & Co., 1920.

with postoperative gouty arthritis. Three of these were observed in the surgical service of Kings County Hospital, Brooklyn, from 1939 to 1944. Another postoperative patient with gouty arthritis was seen in November 1944 at the Lahey Clinic. A survey of the past records at the Lahey Clinic revealed another instance of this complication in 1937.

REPORT OF A CASE

The patient was a 67 year old white man who was seen at the Lahey Clinic on Oct. 23, 1944. He presented a two year history of epigastric pain following meals. During the previous two years he lost his appetite for all types of meat. His diet consisted mainly of milk, liquids, such as clear broth, and mashed potatoes. His pains usually were relieved by aluminum hydroxide solutions. A month before he was seen at the clinic his pains became worse and were not relieved by any medication except morphine administered by his family physician. Gastrointestinal studies on Oct. 19, 1944 demonstrated a gastric neoplasm. During the preceding month he lost 20 pounds (9.1 Kg.).

Physical examination revealed an elderly man with evidence of moderate loss of weight. His weight was 126 pounds (57.2 Kg.), his blood pressure 120 systolic and 70 diastolic and his pulse rate 88. Examination of the head and neck revealed nothing significant, except for the presence of dentures. In the chest no Virchow node was found, and the lungs were clear to percussion and auscultation. No cardiac thrills and no murmurs were heard. The abdomen was scaphoid; slight tenderness was present in the midepigastrium. The liver and spleen were not felt and no masses were palpable. Clearly marked osteoarthritic changes were evident in both the hands and the feet. Digital examination of the rectum failed to demonstrate a rectal shelf or any other unusual condition.

On October 26, the patient was operated on for an adenocarcinoma of the stomach without involvement of lymph nodes. A subtotal Hoffmeister antecolic gastric resection was performed. On his third postoperative day the patient tried to get out of bed. A slight dehiscence of the wound resulted, which did not necessitate immediate closure. In addition to this, atelectasis of the lower lobe of the right lung with pneumonitis developed. On the thirteenth postoperative day he complained of pain in the large toe of his left foot. The toe was hot and tender to palpation and presented the picture of typical gouty arthritis. Laboratory studies on that day showed a serum uric acid level of 5.9 mg. per hundred cubic centimeters; the plasma protein level was 5 Gm. per hundred cubic centimeters. Urinalysis revealed a 3+ reaction for albumin.

The patient responded to treatment and was discharged improved on Nov. 16, 1944.

COMMENT

Gout is a disease entity usually seen in elderly patients. The term "gout" has been employed to signify gouty arthritis and metabolic gout. These entities should be distinguished from each other. Metabolic gout implies merely an increase of uric acid in body fluids (exclusive of hyperuricemia secondary to renal failure). An increased urate concentration, such as this, may exist from birth. Hyperuricemia itself produces no symptoms.[3] However, when urates become deposited on the articular cartilages of joints, symptoms then become manifest. When distress in a joint does occur, then gouty arthritis really exists.

Gouty arthritis as a postoperative complication is infrequently seen. Although its presence may seem to be a minor complication, its underlying significance may not be sufficiently appreciated. This arthritic manifestation should indicate a disturbed protein metabolism. It is a warning that hypoproteinemia is present, and protein depletion may result unless a positive protein balance is reestablished.

The postoperative appearance of gouty arthritis suggests that more organs are involved than appear on superficial examination. The absence of an adequate intake of food depletes the protein and glycogen reserve of the body. The liver supplies the needed glycogen, and muscle tissue gives forth the demanded protein. When the liver exhausts its necessary glycogen, hepatic physiologic processes are impaired. Since it is believed that destruction of uric acid is essentially a hepatic function, it can be appreciated that destruction of urates is limited in the presence of disturbed hepatic physiologic processes.[2] In this way a vicious cycle is accentuated when it is known that protein intake accelerates the excretion of uric acid and lowers its level in the blood.[2] In the surgical patient who is receiving no protein, the stimulus to uric acid excretion is removed and the hyperuricemia is further increased.

What is the effect of hyperuricemia on the kidneys and articulating cartilages of joints? In cases of renal failure it is known that uric acid is one of the first nitrogenous substances to rise. This fact recalls the unknown hepatorenal relationship. The exact nature of this association is not definitely known, although its existence is accepted by surgeons. It can be postulated that in the situation under discussion the hepatorenal relationship is further manifested in uric acid metabolism. The faulty destruction of uric acid by the liver may influence the excretion of uric acid by the kidneys. This retention of uric acid is thus not due to renal failure in the usual

3. Folin, O.; Berglund, H., and Derick, C.: Uric Acid Problem: Experimental Study on Animals and Man, Including Gouty Subjects, J. Biol. Chem. **60**:361 (June) 1924.

accepted use of the term but is more closely allied to the mechanism of the hepatorenal syndrome which has been an occasional complication following surgical procedures on the biliary tract. Such faulty excretion of urates by the kidneys is another factor which further increases the hyperuricemia.

Hyperuricemia is the precipitating agent for the articular manifestations in gouty arthritis. However the pathologic changes in the joints cannot be attributed exclusively to the precipitating agent. A preexisting articular disease, osteoarthritis, must be incriminated as supplying the foundation for the deposition of the sodium salt of uric acid. Past studies have revealed that the formation of tophi cannot be attributed solely to uric acid saturation of the blood.[4] This is believed because the uric acid concentration never reaches the limit of its solubility. Further proof is offered by the fact that in nephritis and leukemia a pronounced hyperuricemia may occur without production of tophi.[4] Thus the importance of a local process, such as osteoarthritis, is stressed for the vicarious excretion of uric acid in the form of crystals of the sodium salt.

The crystalline deposits occur on the cartilaginous joint surfaces of the large toes and fingers. During an acute attack the joint becomes inflamed and tender. After the attack, symptoms subside promptly. If no previous deformity was present, no residual distress persists. Soft tissue swelling may remain for several days. Superficial desquamation of the skin may occur after subsidence of articular signs.

The treatment of this complication involves the increasing of the exogenous protein. A return to normal protein balance eliminates the need for specific medication.

Our survey of 5 instances of postoperative gouty arthritis substantiates the previously mentioned postulates. In every instance the patients were over 55 years of age. All had some degree of osteoarthritis. In all of them hypoproteinemia was displayed with the exception of 1 for whom protein was not determined. Varying degrees of hyperuricemia were present in every patient. No serious residual articular distress was recorded.

A definite physiologic pattern in the production of postoperative gouty arthritis cannot be stated without dispute. Nevertheless, it can be postulated that in the presence of a hypoproteinemia endogenous purine metabolism is accelerated. This accelerated metabolism results in a hyperuricemia, which is the precipitating agent for the deposition of urate crystals on a previously damaged cartilaginous joint surface. When glycogenolysis produces an impairment of hepatic function, the hyperuricemia is further increased by a diminution in destruction of uric acid. If a hepatorenal disturbance becomes evident, as displayed by oliguria, excretion of uric acid is further diminished, with a resultant higher concentration of blood urate. The fundamental cause for this diffuse metabolic disturbance can be attributed to a lowering of the normal blood protein level. This lowered protein level may find its cause either in an inadequate dietary protein intake or in excessive loss of body protein without replenishment. Large amounts of protein may be lost from draining wounds, extensive burns, peritonitis or empyema. A frequent source of protein loss easily overlooked is long-standing albuminuria (case 5). Therefore, protein loss without an adequate protein intake offers an excellent opportunity for the development of postoperative gouty arthritis.

4. Benedict, S. R.: Uric Acid in Its Relations to Metabolism, in Harvey Lectures, 1915-1916, Philadelphia, Williams & Wilkins Company, 1916, vol. 11, p. 346.

SUMMARY

Five patients were observed with gouty arthritis following major surgical procedures. Four patients had cancer, 3 of them having gastric carcinoma. Every patient was a man over 55 years of age. All had some degree of osteoarthritis.

Manifestations referable to the joints usually occurred within five days after operation. The joint of the large toe was the site most frequently involved. Spontaneous remission without residual articular distress occurred.

Determinations of blood chemistry indicated a true hypoproteinemia in 4 patients; a determination was not made for 1. Hyperuricemia of variable degree was recorded in all 5 patients.

The postulated mechanism for postoperative gouty arthritis is hypoproteinemia stimulating endogenous purine metabolism, resulting in hyperuricemia. This hyperuricemia is the precipitating agent for the formation of tophi on an osteoarthritic joint.

This study accentuates the value of an adequate intake of protein for surgical patients. The correction of the hypoproteinemia results in a disappearance of articular symptoms.

CONCLUSION

The facility with which protein depletion occurs can be appreciated when one considers that a surgical patient usually is given a diet

with little or no protein. This may be the fault of the surgeon, who may limit the preoperative diet, especially when surgical procedures on the gastrointestinal tract are contemplated. The patient's poor appetite may be another contributing factor.

During the postoperative period the disturbance may not be corrected. Usually only a liquid diet is permitted for several postoperative days. When the patient does not eat or when vomiting occurs, protein starvation is further increased because the usual intravenous medicaments contain only dextrose and isotonic solution of sodium chloride. This protein deficiency is the fundamental basis for the development of postoperative gouty arthritis. Therefore, the appearance of gouty arthritis in a surgical patient should suggest a more serious metabolic discord than superficial examination would indicate. Although serious, this complication can be easily rectified by establishment of a positive blood protein balance.

Dr. John F. Raycroft granted permission to present the clinical and laboratory data of the 3 cases observed in his surgical service at King's County Hospital, Brooklyn.

ACRYLIC RESIN AS AN IMPLANT FOR CORRECTION OF FACIAL DEFORMITIES

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Implants, foreign or otherwise, have their place in the reconstructive field of plastic surgery. In the past, the materials used as implants were chiefly bone or cartilage. Their use dates back to about 1861, when rib grafts were made to supply the skeletal framework of a reconstructed nose. Other materials that have been used include bovine cartilage, ear, rib and septal (autogenous) cartilage and homocartilage.

Bovine cartilage has been used in place of an autoplastic transplant but with little success. Ear cartilage is often used and with definite success in cases of minor repair, where only a small defect exists. Homocartilage [1] as a material is fairly satisfactory but not as satisfactory as autogenous cartilage. Homocartilage can be obtained at autopsy, sterilized and stored, to be used as needed. This type has the advantage of eliminating the somewhat hazardous operation of costal resection, with its prolonged incapacity and morbidity. Regardless of the cartilage used, there is the ever present possibility that the graft may curl, although curling is less likely if transverse and longitudinal crevices are cut on the under side of the implanted costal cartilage.

In 1861 Ollier reported the transplantation of living bone for the construction of a nasal bridge.[2] Hardi transplanted the first and second phalanx of a finger but with poor results. Koenig advocated the use of the outer table of the frontal bone together with its overlapping skin for the building of a saddle nose. Israel transplanted a free bone graft taken from the anterior surface of the tibia.[3] This graft was successful, and it inaugurated a new era in the use of free transplants.

Comparison of bone with cartilage for free transplants shows that each has its place. For reconstruction of a saddle nose, for instance, bone is not as satisfactory as cartilage, as often absorption of the bone implant takes place. It has long been known that unless bone is placed in contact with bone or made to perform its weight-bearing function it often is absorbed in a comparatively short time. Ollier claimed that the presence of periosteum is essential for survival of bone. Others are of the opinion that periosteum does not play an essential part, although its presence may have some bearing on the rapidity of regeneration. Davis [4] has expressed the opinion that a transplant of bone becomes absorbed even if it is in contact with living bone.

Ivory is another material that has been used.[5] Gluck, of Paris, in 1884 was the pioneer in the employment of this material. He used it successfully as a foreign implant to bridge fractures of long bones. Jacques Joseph, of Berlin, was the first to use ivory for the correction of a saddle nose of both traumatic and syphilitic origin.[3] The chief difficulty with ivory is the fact that it is difficult to prepare and shape prior to its insertion.

ACRYLIC RESIN

Acrylic resin, a polymerized methyl methacrylate, is a synthetic material used widely in American dentistry since 1939.[6] It is a stable, clear material slightly heavier than water.[7] This

Owing to lack of space, this paper has been abbreviated for publication in THE ARCHIVES by omission of a number of the illustrations. The complete article will appear in the author's reprints.

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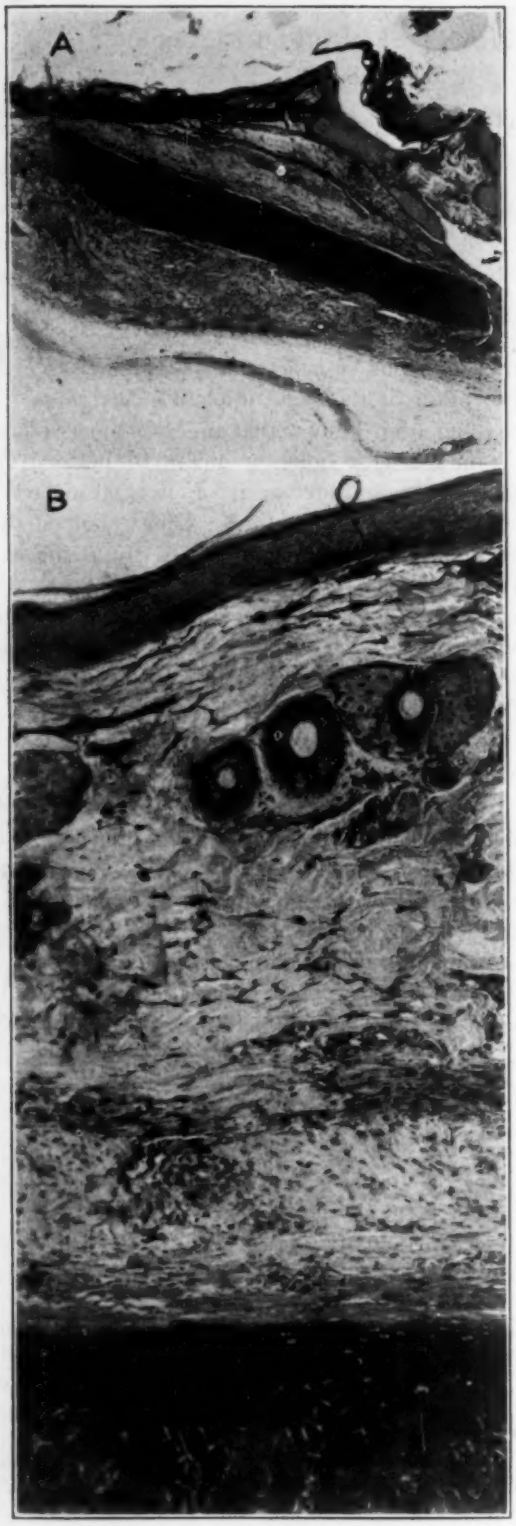

Fig. 1.—Ear cartilage implant. *A*, low power (× 20), showing moderate degree of uniform infiltration about the implant; *B*, high power (× 135), showing the type of infiltration.

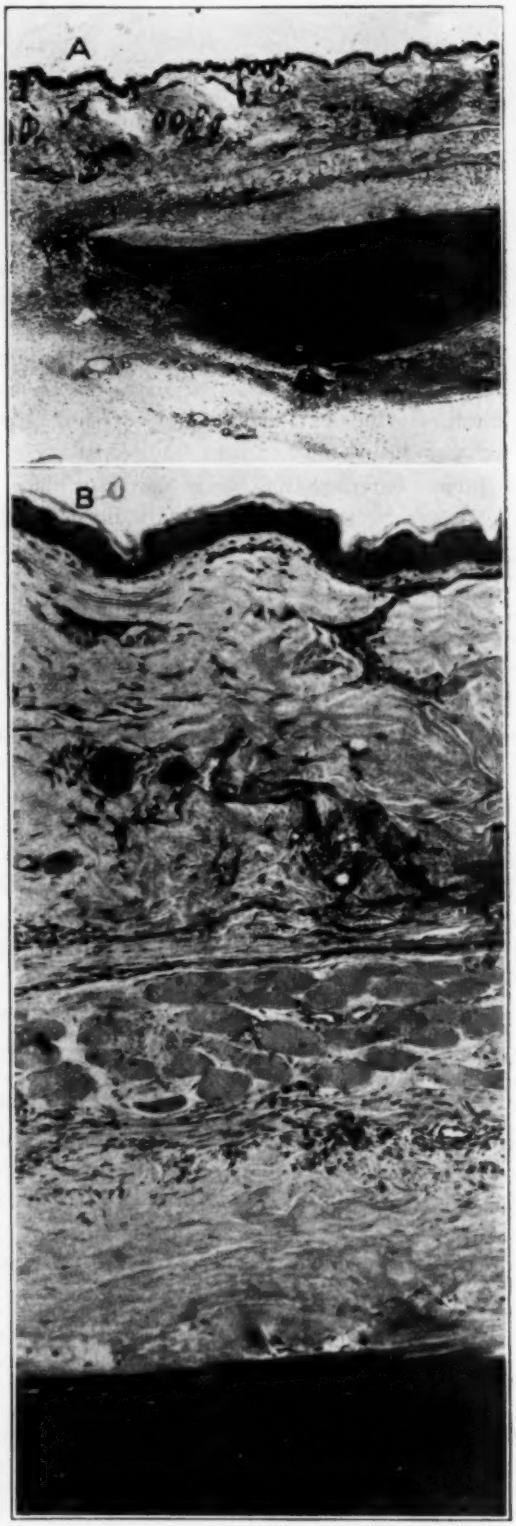

Fig. 2.—Costal cartilage implant. *A*, low power (× 20), showing more infiltration on the inferior surface of the graft; *B*, high power (× 102), showing type of infiltration.

Fig. 3.—Ivory implant. *A*, low power (× 19), showing more abundant infiltration about portions of the graft (ivory removed); *B*, high power (× 130), showing type of infiltration.

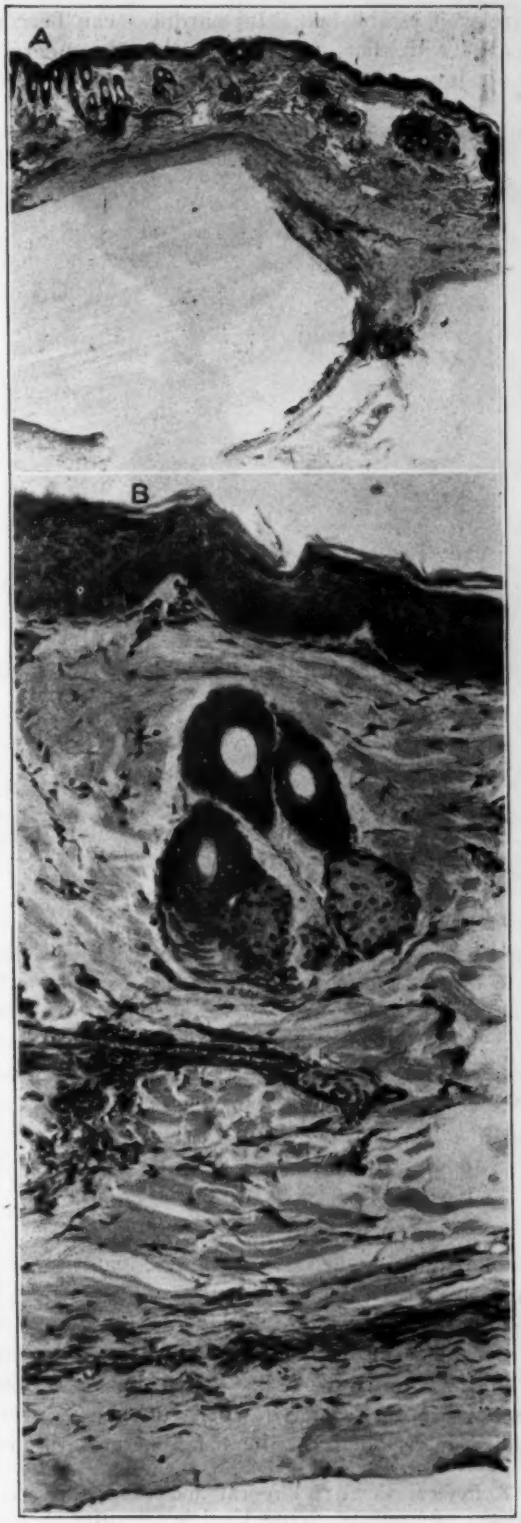

Fig. 4.—Acrylic resin implant. *A*, low power (× 20), showing slight infiltration about the periphery of the implant (resin removed); *B*, high power (× 174), showing area of slight infiltration (bottom).

material had its origin in Germany through the research efforts of Rohn in 1900. The chemical nature and general properties of acrylic resin are well established. Its hardness can be compared with that of 24 carat gold. It undergoes no significant change when boiled. Acrylic

Fig. 5.—Deformity of the chin of a 23 year old woman, due to bilateral ankylosis and surgically corrected in 1936. At the left, the patient is wearing an acrylic resin implant inserted at will in the labial sulcus, which was previously lined with epithelium from a thigh. At the right is the cast of the face before correction.

resin has a water solubility of about 0.1 per cent when placed in distilled water for one week.[8] Alcohol and ether have no visible effect on the material, although acetone and chloroform produce a noticeable softening. Acrylic resin has a tensile strength of from 9,000 to 12,000 pounds (4,080 to 5,440 Kg.) per square inch (6.5 sq. cm.).[9] It will, however, soften when submitted to a temperature of about 200 F.

FOREIGN BODY REACTION

The question arises, Does this material produce any foreign body reaction when buried in living tissues? Davis,[9] in his report on the use of acrylic resin as a helmet for protection against craniocerebral injuries, has stated that no serious tissue reaction occurred when such pieces were buried by accident in the brain or soft tissues. Another question is, How does the reaction, if any, compare with the reaction set up when known satisfactory grafts are implanted?

Experimental Studies.—To arrive at an answer, clinically as well as microscopically, pieces of known satisfactory grafts were implanted under the skin of a monkey. These, together with a piece of acrylic resin, were implanted under careful aseptic conditions. These grafts, together with the surrounding tissue, were removed after seven days for microscopic examination.

In incision 1 ear cartilage (autogenous) was implanted; in incision 2 costal cartilage (autogenous) was implanted; in incision 3 ivory was implanted, and in incision 4 acrylic resin was implanted. The incisions in the skin were closed with silk, and a sterile dressing was applied. This dressing was covered with adhesive tape on a widely shaved area on the back of the animal, where contamination and accessibility were least likely to occur.

Summarization of Histologic Sections.—From this limited series of trials one can visualize the tissue reactions caused by the various known

Fig. 7.—Congenital underdevelopment of the mandible.

satisfactory implants and compare them with those caused by the acrylic resin. One might also conclude that acrylic resin produced less inflammatory infiltration in the early stages and yet a satisfactory fibrosis. In all grafts there was little change in the true skin, and this is

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especially true of the acrylic resin, around which the fibrosis is confined to the immediate area of the implant.

METHOD OF PREPARING AN ACRYLIC IMPLANT FOR CORRECTION OF FACIAL DEFECT

An impression may be taken of the face, including all parts necessary to get proper proportions. This impression may be taken with Negocoll (a colloidal material), other colloid substance or plaster of paris. The eyelashes and eyebrows are best covered with petrolatum, over which a flat piece of wet cotton is applied to prevent the material from adhering to the hair and getting into the eyes. The nasal cavity is packed lightly with a small mass of moist cotton around small tubes to facilitate breathing.

From the negative a positive is prepared, of plaster, wax, Hominit, stone or metal as desired. On this positive a wax mold of the

Fig. 8.—Same patient as in figure 3 four months after implantation of acrylic resin through an external incision under the chin. The implant is buried under the soft tissues over the symphysis of the mandible.

defect is prepared. This is also tried to the defect itself. In the case of a saddle nose, the wax should be longer than the cast to allow compensation for the soft tissues over the globula of the nose. The under surface is concave and the lateral sides free from sharp edges. "Pink wax" (dental) is satisfactory. It can be molded by flame or softened in warm water. The wax pattern should be made slightly larger than is required, as one can easily reduce the size by means of surgical burs. Perfect molding of the wax to a cast is not necessary, and often the

Fig. 10.—Loss of the nasal bridge from traumatic injury (industrial).

mold is too small, as the implant is not to be inserted on the skin as the impression recorded. The implant is to fit over the bone, under the skin and soft tissues. A visual impression of the bony surface on which the implant is to lie is much more important. It is on this palpable hard surface that the implant must lie. Often I take no impression unless one is desired for a record. The general bony contour is noted, and the wax is prepared with the idea that it is to be inserted under the skin and subcutaneous tissues, not to be laid on the external visual defect.

The wax pattern, prepared in any manner desired, is now ready to be reproduced in acrylic resin. This is done by investing the wax in plaster of paris in a metal flask, the flask separated and the wax removed with boiling water. This leaves a mold in plaster the exact size and shape of the original wax pattern. The acrylic resin, purchased in the form of a powder and a liquid, is ready to be mixed. For this a small, clean jar can be used. The liquid, usually methyl methacrylate, is mixed with the powder to form a doughlike mass. When this consistency is

obtained, the material is packed firmly in the prepared mold slightly in excess and covered with cellophane or tinfoil. The flask is closed, clamped and placed in boiling water (100 C.) for one hour for polymerization. After boiling, it is allowed to cool and is separated. The soft, putty-like mass is now hard and can be removed in toto by cutting away the plaster.

Polishing is a not too difficult procedure. A large surgical bur can be used to remove whatever excess appears. On the under surface many cone-shaped openings are made, but these should not extend through the material. The outer surface, especially of a nasal implant over which the skin is thin, is left smooth but not glazed.

Fig. 11.—Same patient as in figure 10. Profile showing the nasal bridge after implantation of acrylic resin. The slight hump on the bridge is due to scar mass.

The acrylic resin implant is now ready to be cleaned and sterilized. Green soap and a hand brush will remove all debris; cleansing should be carefully done, especially on the under surface, where the holes are made. It is placed again into liquid soap and then immersed in boiling water for a few seconds, which is adequate sterilization. When removed from the boiling water, the implant is best kept or stored in a sterile wide-mouthed glass bottle. In the operating room the glass bottle with the acrylic resin might be autoclaved, but this is a questionable procedure and an unnecessary precaution. A satisfactory last minute sterilization can be done by merely removing the top of the bottle and pouring in sufficient 95 proof alcohol to cover the implant.

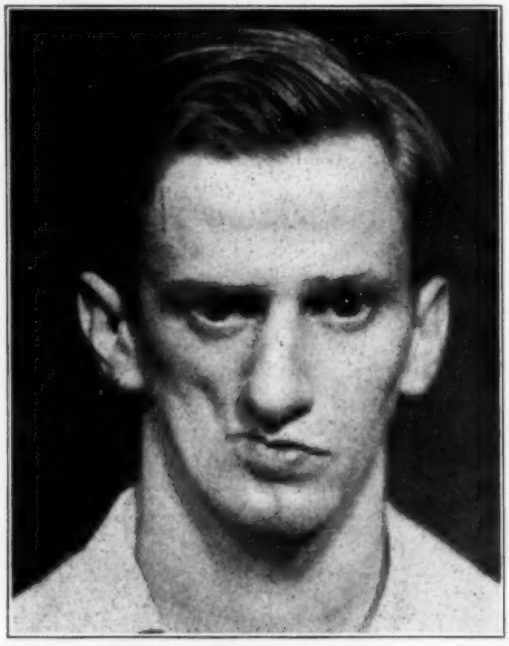

Fig. 14.—Underdevelopment of the right side of the face (mandible and maxilla) caused by a traumatic injury when the patient was 6 years old.

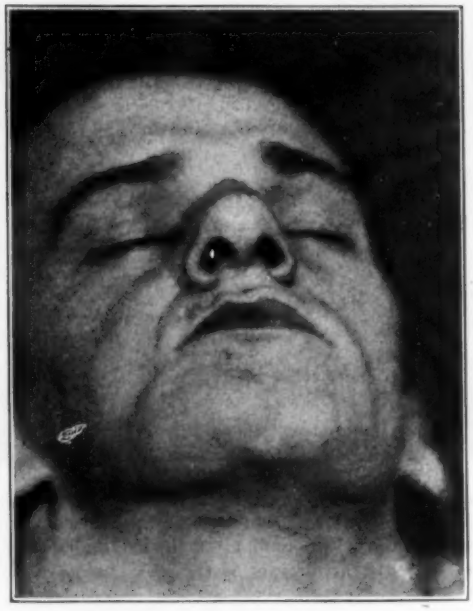

Fig. 15.—Same patient as in figure 14 after correction of the deformity of the mandible with an acrylic resin implant (overcorrected to compensate for the lack of facial development). The depression of the maxilla was corrected with an intraoral skin graft and a dental restoration of acrylic resin.

IMPLANTING THE ACRYLIC RESIN

The surgical field is prepared as one would prepare it for the reception of any implant (bone or cartilage). A precaution worthy of mention is ligation of all bleeding vessels before the implant is made. When one prepares a cavity over the symphysis of the mandible, for instance, the vessels at the mental foramen should be avoided if possible. In such a location one may not be able to stop the bleeding by a pressure bandage, as this is a hard substance and it may not conform perfectly to the bone to stop bleeding by direct pressure. The periosteum is not disturbed.

The cavity prepared, the implant is removed from the alcohol by means of a sterile tooth forceps and held in an ether bath for a few seconds. The forceps, still clasping the resin, is passed through air rather than dried with sterile gauze. After the implant has been placed in the position desired and the wound has been closed in the usual manner, a light pressure bandage should be applied. Petrolatum gauze over the skin and moist cotton will usually suffice. This may be held in position by adhesive tape or bandage. An uneven pressure bandage is objectionable, as it may dislodge the graft.

After twenty-four hours the bandage should be removed and the graft palpated to determine whether it has remained in proper position. If not, it is possible to manipulate it slightly. A gentle pressure bandage should be reapplied and changed when necessary for a period of five to six days.

Clinical observations of the skin are surprisingly gratifying. Very little redness is seen, especially when small grafts are implanted. It is known that some persons (a very small percentage) are allergic to acrylic resins worn as a dental plate. For this reason, it is necessary to prepare the patient for such a possibility. One could test the patient for such a reaction before the operation, but in my experience no reactions have occurred after the implantation. From my observation, there is less cutaneous reaction with acrylic resin than with cartilage, especially homogeneous cartilage.

PRECAUTION

I deem it advisable to instruct patients before and after operation that such implants may produce inflammatory changes at some future date. In the event that such noticeable reactions occur, they are advised to have the grafts removed. In my limited experience with this material for over a period of about three years, no person has had any untoward clinical effects. Not until such an implant has been embedded for ten or twenty years is one prepared to state its final outcome. Further investigation is warranted, as there are unlimited possibilities for use of this material in reconstructive surgery. It may be advisable to bury metal in the acrylic resin implant, so that in case of future traumatic injuries the graft will be radiopaque to roentgen rays.

ANOMALOUS FUSION OF THE SCAPHOID AND THE GREATER MULTANGULAR BONE

LIEUTENANT COMMANDER M. G. HENRY (MC), U.S.N.R.

Congenital fusion of carpal or tarsal bones is a rare anomaly. It may occur independently but usually is associated with synostosis of some of the interphalangeal joints. There are amazingly few English and American papers on the subject.

Foreign writers offer varied arguments for their individual theories regarding the cause of this anomaly. I believe that one should accept only the scientific anatomic facts which tend to bear out the theory of arrested or defective development. These anatomic facts show that ossification centers appear in different years for each of the carpal bones except the greater multangular and the scaphoid bone, both of which show ossification centers in the sixth year of life. Because of this embryonic similarity, it seems logical that fusion of bones in the wrist would most probably involve those whose bony growth occurred at identical periods of development.

Rochlin [1] in 1928 reported symmetric fusion of some of the carpal bones associated with hypoplasia of the finger joints.

Kewesch [2] in 1934 expressed the opinion that the same developmental disturbance which produces synostosis of interphalangeal joints also causes fusion of carpal bones. He reported a case of symmetric synostosis of carpal and tarsal bones in 3 members of the same family.

Both Kewesch [2] and Esau [3] in 1933 pointed out that the symmetric distribution of fusion of the carpal and tarsal bones and the frequency with which the anomaly is associated with aplasia of the interphalangeal joints tend to indicate a congenital and hereditary character of the anomaly.

Mestern [4] in 1934 reported bilateral fusion of carpal and tarsal bones occurring together in 2 families.

Familial tendencies were observed by Cushing [5] in 1916. He stated that it is probable that the trait may be transmitted in its most outspoken form by a parent who appears to be but slightly affected but never through parents neither of whom show evidence of the anomaly. He studied 72 families, or 302 persons, and found the defect in 25.8 per cent. Of 150 children of parents who had evidence of fusion, 78 of them, or 52 per cent, were affected. Cushing stated the belief that the ankylosing process may be caused by an incomplete prenatal development of the joint.

The following case report concerns a patient who was examined at a military base because of an injury to his wrist. Roentgenograms showed not only fracture of the scaphoid but the interesting and rare anomalous fusion of the scaphoid with the greater multangular bone.

BRIEF REPORT OF A CASE

R. B., a man aged 25, fell on his extended hand during commando drill and felt something crack in his wrist. Examination showed the pathologic process to be limited to the wrist, which was swollen and tender; there was much pain on extension, and local severe pain on pressure over the radial aspect of the carpus. Extension of the thumb was limited and painful. Roentgenograms showed simple fracture of the inner third of the scaphoid, which was congenitally fused completely with the greater multangular bone. There was no past history of injury to this wrist. No other anomalies were present, and none were known to exist in members of the immediate family. The anomaly was limited to the one wrist. There was no aplasia of the interphalangeal joints.

Most authors agree that the underlying cause of synostosis of carpal or tarsal bones is absence of or imperfect differentiation of the intermediary

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This article has been released for publication by the Division of Publications of the Bureau of Medicine and Surgery of the United States Navy. The opinions and views set forth in this article are those of the writer and are not to be construed as reflecting the policies of the Navy Department.

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zone in the development of the ossification center. It can be accepted that fusion of such bones is a result of hypoplasia or aplasia of the individual articulations.

It has been proved that the hereditary trait for this anomaly behaves as a unit character; it

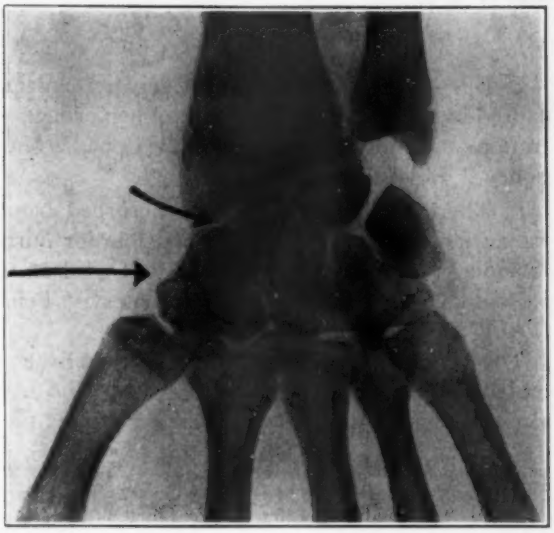

Fig. 1.—Fusion of the greater multangular and the scaphoid bone with fracture of the inner third of the scaphoid.

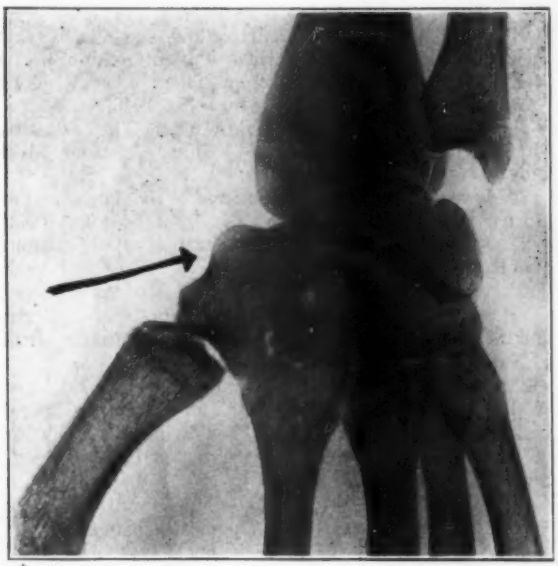

Fig. 2.—Complete fusion involving both rows of carpal bones may account for limitation in motion.

is transmitted in accordance with the mendelian law as a dominant factor and is not sex linked.

The patient had permanent limited motion in the wrist. It is impossible to state whether the persistent limitation of motion was inherently caused by the fusion of the two bones or whether it was due to fracture of the scaphoid, which fortunately had not destroyed the nutrient artery and so healed without the usual difficulties.

SUMMARY

A case of congenital fusion of the greater multangular and the scaphoid carpal bone was discovered on roentgenographic examination of an injured wrist.

Generally fusion of the carpal bones is associated with synostosis of some of the interphalangeal joints.

Fusion of carpal or tarsal bones is hereditary, and the trait is transmitted according to the mendelian law as a dominant factor which is not sex linked.

The greater multangular bone and the scaphoid are the only two carpal bones to have identical times for the appearance of their ossification centers; this should lend argument to the theory of arrested development as the cause of this rare anomaly.

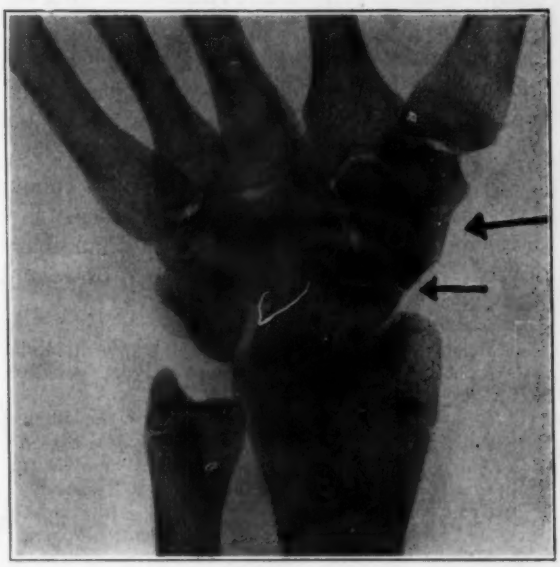

Fig. 3.—Fracture of the inner third of the scaphoid bone with no dislocation heals in normal fashion and does not produce the disabling and painful wrist which commonly results from fracture of the middle third of the scaphoid with nonunion and injury of the nutrient artery (Henry, M. G.: Fractures of the Carpal Scaphoid Bone in Industry and in the Military Service, Arch. Surg. **48**:278 [April] 1944).

DEGENERATIVE WHITE BLOOD CELL PICTURE AS AN INDICATION OF TOXEMIA FROM BURNS

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SYRACUSE, N. Y.

In spite of the enormous amount of work that has been done on the subject, the question of a specific toxemia in burns is not yet settled. There are at least six distinct attitudes to be found in the literature:

1. There is no true burn toxemia (Fender,[1] Lam [2]).

2. Infection can account for the whole picture of burn toxemia (Aldrich,[3] Cruickshank,[4] Marsh,[5] McSwain,[6] Wakeley,[7] Siler [8]).

3. Hemoconcentration (secondary shock) can account for the whole picture of burn toxemia (Underhill and Kapsinow,[9] Osterberg and others [10]).

4. Whatever burn toxemia really is, it cannot be explained solely on the basis of plasma loss and hemoconcentration (McClure,[11] Elkington,[12] Boyce,[13] Harkins,[14] Lee and Rhoads,[15] Bosse and associates [16]).

5. Burn toxemia is a result of hepatic damage caused by anoxia, in turn a result of hemoconcentration and impaired circulation (Cope,[17] Mahoney,[18] Boyce [13]).

6. There is a true burn toxin absorbed from the surface of the burn and responsible for burn toxemia (Robertson and Boyd,[19] Davidson,[20] Rosenthal,[21] Wilson,[22] Blalock,[23] Tenery,[24] Penberthy,[25] Elman,[26] Douglas [27]).

The period of toxemia has been generally accepted as coming after secondary shock and before the onset of infection.[28] Wilson [22] has set the

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26. Elman, R.: Early Mortality of Burns as Influenced by Rapid Tanning and by Transfusions, Ann. Surg. **117**:327-331 (March) 1943.

27. Douglas, B.: The Treatment of Burns and Other Extensive Wounds with Special Emphasis on the Transparent Jacket System, Surgery **15**:96-143 (Jan.) 1944.

28. Harkins, H. N.: The Treatment of Burns, Springfield, Ill., Charles C Thomas, Publisher, 1942, pp. 38, 70, 79, 134, 202 and 213.

phases of burns more accurately as follows: (1) primary shock, up to two hours; (2) secondary shock, two to twenty-four hours; (3) acute toxemia, six to one hundred hours, and (4) septic toxemia, after one hundred hours.

Primary shock, which is neurogenic in origin, is usually considered of infrequent importance, though it may be held responsible for a small proportion of deaths (2.5 per cent).[22] It is rarely observed in men of the fighting services unless other injuries are present in addition to the burns, but it is seen in marked degree in women and children and old people who have been removed from burning and bombed houses.[29]

Secondary shock is a result of failure of circulation in the presence of hemoconcentration and falling blood pressure.[30] The symptoms and signs can be explained entirely on the basis of plasma loss.[31] These are rising pulse and respiratory rates, pallor, thirst, anxious restlessness and falling blood pressure.[30] The temperature is subnormal.[22] Secondary shock may appear within one hour of the injury, but as a rule there is a relatively safe period in the first six hours in which the patient, though severely burned, can stand a reasonably long journey.[30] At the end of six hours one half of the hemoconcentration likely to occur is present,[24] the peak being passed by the end of forty hours.[32] If the concentration of the blood does not return to near normal within seventy-two hours, the patient dies.[28] Deaths due to secondary shock have been estimated as occurring in as high as 60 and even 80 per cent of cases of fatal burns.[33]

Acute toxemia (toxic shock) is that phase of a burn characterized by drowsiness or semicoma, high fever, rapid pulse, rising nonprotein nitrogen level of the blood and diminished urinary output.[18] The blood pressure is lowered as in secondary shock.[22] There is frequently nervousness marked by restlessness, vomiting, hiccup and convulsions.[27] During the toxic period there are disturbances in hepatic function,[34] and jaundice may be present.[28] Wilson believes that for 13 deaths in 65 severely burned children he eliminated all causes but "acute toxemia."[22] Boyce found that of 217 fatal burns 36 were associated with uncomplicated, and 92 with complicated, toxicity.[13]

Deaths occurring after this toxic phase are due usually to either septic complications or parenchymatous organ damage or result from the presence of large granulating surfaces. The last complication may be so severe that emergency skin grafting is at times necessary, especially in children.

Secondary shock and acute toxemia are difficult to separate clinically because secondary shock, whether treated or not, carries over into the toxic phase and both may be affected by the presence of sepsis or other complications. In addition, while secondary shock is fairly constant and predictable and can be estimated by the hematocrit, toxemia is erratic and uncertain[22] and has as yet no corresponding laboratory procedure to predict its development.[18] It is with a view to offering a largely neglected aspect of the white blood cell picture as such a needed laboratory procedure that this work is presented.

HISTORICAL SUMMARY

The first mention of the white blood cells in burns was made in the eighteen sixties by Wertheim.[35] While studying the changes of the red cells of experimentally burned dogs, he noted the presence of "many white corpuscles."

Near the turn of the century the white cells in human burns were studied by Hock,[36] Bardeen,[37] Dohrn[38] and Locke.[39] All found a pronounced leukocytosis immediately following the burns, Locke reporting some extremely high figures. In Locke's series of 10 cases, 6 of which were fatal, there was a white cell count of 73,000 per cubic millimeter in 1 of the fatal cases thirty minutes after the burn. In the other 5 fatal cases the counts varied from 30,000 to 51,000 per cubic millimeter thirty minutes to two hours after the

29. Wakeley, C. P. G.: War Burns and Their Treatment, Practitioner **146**:27-37 (Jan.) 1941.

30. McIndoe, A. H.: First-Aid Treatment of Burns, Lancet **2**:377-378 (Sept. 27) 1941.

31. Beard, J. W., and Blalock, A.: Experimental Shock: VIII. The Composition of the Fluid That Escapes from the Blood Stream After Mild Trauma to an Extremity, After Trauma to the Intestines and After Burns, Arch. Surg. **22**:617-625 (April) 1931.

32. Elkington, J. R.; Wolff, W. A., and Lee, W. E.: Plasma Transfusion in the Treatment of the Fluid Shift in Severe Burns, Ann. Surg. **112**:150-157 (July) 1940. Tenery.[24]

33. McIndoe.[30] Wakeley.[29]

34. Wolff, W. A.; Elkington, J. R., and Rhoads, J. E.: Liver Damage and Dextrose Tolerance in Severe Burns, Ann. Surg. **112**:158-160 (July) 1940.

35. Wertheim, G.: Ueber Verbrennung und Verbrühung, Med. Jahrb. **16**:36-45, 1868.

36. Hock, A.: Ueber Pathogenese des Verbrennungstodes, Wien. med. Wchnschr. **43**:737-741 (April 22) 1893.

37. Bardeen, C. R.: A Review of the Pathology of Superficial Burns, with a Contribution to Our Knowledge of the Pathological Changes in the Organs of Rapidly Fatal Burns, Johns Hopkins Hosp. Rep. **7**:138-179, 1898.

38. Dohrn, K.: Zur pathologischen Anatomie des Fruehtodes nach Hautverbrennungen, Deutsche Ztschr. f. Chir. **60**:469-499, 1901.

39. Locke, E. A.: A Report of the Blood Examinations in Ten Cases of Severe Burns of the Skin, Boston M. & S. J. **147**:480-484 (Oct. 30) 1902.

burn. Locke concluded that in fatal cases the count is usually 50,000 per cubic millimeter or above.

Neither Hock, Bardeen nor Locke reported counts made more than twenty-two and one-half hours after the burn. Dohrn,[38] however, studied the counts longer and found "a considerable increase in white blood cells up to about the sixth hour, after which a gradual decrease followed, which in the course of two days restored the number of white cells nearly to normal."

While Hock and Bardeen noted the leukocytosis as chiefly polymorphonuclear, Locke found the percentage of neutrophils to be "not so much above normal as in the inflammatory leukocytoses." He also found evidence of degeneration in nearly all specimens, in 1 case involving nearly 50 per cent of the cells. In 4 of the 6 fatal cases Locke noted "lilac-colored granules to be very abundant" in the cytoplasm. Myelocytes were present in 2 cases.

Pfeiffer,[40] in 1913, made an important contribution regarding the total white cell count. He studied the leukocytes in experimentally burned guinea pigs and found three main types of reactions: (1) in the ones with the severest burns, a primary leukopenia; (2) in those with severe fatal burns, a primary leukocytosis with secondary leukopenia, and (3) in those with light burns (one eighth to one sixth of the body surface), an intensive leukocytosis with gradual return to normal. Pfeiffer compared this leukopenia with that of anaphylactic shock and considered it due to the absorption of traces of necrotic tissue. Although he cautioned strongly against applying his results to human beings, he advised that the relation be worked out.

Schreiner and Pucsko,[41] in 1925, were the first to associate the fall in the white cell count following severe burns with the onset of the period of toxemia. They stated that they "have frequently been able to observe leukopenia at the time when toxicity was asserting itself, and have learned to appraise it as a threatening symptom." They are apparently the first authors since Locke to find degenerative changes in the differential picture and the first to call attention to the marked neutrophilic shift to the left, which they considered of more prognostic value than the morphologic changes in the cell. Scheiner and Pucsko found that the eosinophils are good indicators in estimating prognosis, that they disappear in severely burned patients and reappear as the patients recover.

In 1933 McIver[42] found the elevated white cell count to be a "constant factor" in 16 cases of human burns. Although he did not discuss the progress of the count, a review of his cases reveals a definite tendency for the white cell count to fall during the period from the second to the sixth day and to return to a higher level thereafter. In 1 case, for example, the count was 24,000 per cubic millimeter on the first day, 8,600 on the fifth day, 13,480 on the sixth day and 23,600 on the seventh day (no counts being made on the second, third and fourth days). In all, the tendency for the count to fall after a burn and to return to a leukocytotic level in the event of survival is seen in 7 of the 16 cases. In 6 of the remaining cases, counts were insufficient, and in 3 the tendency was not apparent.

In 1935 Fasal[43] systematically studied the blood picture in severe burns and noted that a sudden regression often follows the original leukocytosis. This regression occurs together with other toxic changes, such as "a left shift of the neutrophils to the myelocyte and promyelocyte stages, basophilia of the protoplasm, vacuolation and fragmentation of nuclei." Fasal observed these changes as coming on at about the fourth day after the burn and considered their presence as the "strongest possible" indication for immediate blood transfusion.

In 1937 Lambret, Driessens and Cornillot[44] studied the white cell count in experimentally burned rabbits. They found that twelve to twenty-four hours after the burn and after an initial leukocytosis there was an almost complete disappearance of white cells, particularly the neutrophils, such as to cause an actual leukopenia. They offered no explanation for the phenomenon.

In 1938 Wilson and his co-workers[22] found that "a leukocytosis in venous and capillary blood was a frequent, though not invariable, occurrence." In the case of a 3 year old boy with "moderate" scalds, the total white cell count fell from 20,000 per cubic millimeter on the first day to a low point of 6,800 per cubic millimeter on

40. Pfeiffer, H.: Das Problem des Verbrühungstodes, Vienna, E. Hölzel, 1913, pp. 74 and 79.

41. Schreiner, K., and Pucsko, O.: Die Veränderungen des Blutbildes nach Verbrennungen, Med. Klin. **21**:1882-1883 (Dec. 11); 1925-1928 (Dec. 18) 1925.

42. McIver, M. A.: A Study in Extensive Cutaneous Burns, Ann. Surg. **97**:670-681 (May) 1933.

43. Fasal, P.: Ausgedehnte Verbrennung: Indikationsstellung zur Bluttransfusion durch toxische Veränderungen des weissen Blutbildes, Wien. klin. Wchnschr. **48**:282 (March 1) 1935; Veränderungen im weissen Blutbild als Indikator fuer lebensrettende Bluttransfusionen bei Verbrennung mit infauster Prognose, ibid. **48**:1591 (Dec. 20) 1935.

44. Lambret, O.; Driessens, J., and Cornillot, M.: Variations du taux des hématies, des leucocytes et de l'hémoglobine au cours des brûlures expérimentales du lapin, Compt. rend. Soc. de biol. **125**:661-662, 1937.

the fifth. Toxemia was not, however, considered responsible.

Harkins [28] stated in his textbook (1942) that "the significance of leukocytosis is probably merely that it indicates trauma. Later it may indicate infection and a low grade leukocytosis may continue for some time from that cause."

From this historical review the following facts can be drawn about the blood picture in severe burns: (1) that there is a leukocytosis immediately following the burn, which in most cases is directly proportional to the degree of severity; (2) that this leukocytosis often falls between the second and sixth days and may reach an actual leukopenia, returning thereafter to its earlier level of leukocytosis in the event of survival, and (3) that the differential white blood cell picture may show a high nonfilamented cell count, degenerative changes in the cells, absence of eosinophils and presence of myelocytes—the last findings being said to indicate a serious prognosis.

The fall in leukocytes between the second and sixth days is attributed to toxemia by Schreiner and Pucsko [41] and Fasal.[43] McIver,[42] Wilson and co-workers [22] and Harkins [28] all have different explanations. The changes in the differential picture, although recognized as of serious import, have not been satisfactorily explained.

The experimental work on animals, except guinea pigs, bears out the applicability of the results to human beings; in guinea pigs extremely severe burns were found to produce leukopenia without preliminary leukocytosis (Pfeiffer's first type).[40] Whether this ever occurs in human beings I do not know.

REPORT OF CASES

Four previously healthy men were admitted by ambulance from the factory where they were employed to the University Hospital of the Good Shepherd on April 3, 1944. All 4 had been burned by hot oil which sprayed over them when a high pressure line burst, 3 severely and 1 less so. They were similarly treated, with petrolatum gauze dressings under moderate pressure, plasma and dextrose in saline solutions parenterally and sulfadiazine orally.

CASE 1.—R. B., aged 26 years, suffered second and third degree burns covering 85 per cent of the body surface. The hematocrit reading was 50 volumes per cent on admission and 46 volumes per cent on the next day. The patient died on April 9, six days after his admission to the hospital.

Necropsy Observations.—Anatomic: Extensive superficial burns were noted. There were acute bronchitis (probably early bronchopneumonia) and toxic changes in the heart, liver, kidneys and adrenals.

Microscopic: The lungs showed acute bronchitis with severe congestion of the mucous membrane and some hemorrhage and acute bronchopneumonia with rather severe hemorrhage into the alveoli. There was congestion of the spleen, and the liver showed early central necrosis. In the kidneys slight capillary thickening of the glomeruli was observed. The adrenals were congested, with swelling and patchy hydropic degeneration of cortical cells.

CASE 2.—C. G., aged 56 years, suffered second and third degree burns covering 65 per cent of the body surface. The hematocrit reading was 47 volumes per cent on the day of his admission to the hospital and 58 volumes per cent on the next. This patient appeared to be holding his own till the sixth day after the burn, when gangrene developed in the right foot; later, signs of pneumonia were noted in both lungs. He died on April 18, fifteen days after admission.

Necropsy Observations.—Anatomic: There were extensive superficial burns, massive hypostatic pneumonia, multiple thrombosis of vessels, recent infarcts of the heart and spleen, gangrene of the right foot and toxic changes in liver, spleen and kidneys.

Microscopic: Examination of the heart showed arteriosclerosis, multiple small organizing areas of infarction, recent microscopic areas of myocardial degeneration and some fragmentation. The lungs showed massive bronchopneumonia and pulmonary thrombosis. There was recent infarction of the spleen, and the liver gave evidence of slight cholangitis and a few areas of focal necrosis. The kidneys showed chronic pyelonephritis of moderate degree. The adrenals disclosed a few areas of focal necrosis in the cortex, and there was an organizing thrombus in the main artery and branches of the anterior tibial artery. The mediastinal tissue revealed chronic and acute inflammation.

CASE 3.—M. K., aged 45 years, received second and third degree burns of 35 per cent of the body surface. He was discharged from the hospital improved on May 27 but was to return for additional skin grafting.

CASE 4.—C. S., aged 43 years, was admitted with first degree burns covering 35 per cent of the body surface and second and third degree burns covering 5 per cent. He recovered and was discharged on April 29, 1944.

The pertinent laboratory data in each case are given in table 1. As will be seen, the white blood cell counts were not started till the second day after admission in 3 cases and not until the third day in 1 case.

In each instance the differential counts were done by me and at least 200 cells counted from each smear. The percentage of nonfilamented cells is taken as the proportion of nonfilamented neutrophils to total neutrophils and not to total white cells. The myelocytes are included as neutrophils as well as reported in a separate column. Juvenile forms when present are not reported separately. In determining the percentage of nonfilamented cells I have used the oil immersion lens exclusively and have adhered to the criteria laid down by Cooke and Ponder [45];

45. Cooke, W. E., and Ponder, E.: The Polynuclear Count, London, Charles Griffin & Co., 1927; cited by Farley, D. L.; St. Clair, H., and Reisinger, J. A.: The Normal Filament and Nonfilament Polymorphonuclear Count: Its Practical Value as a Diagnostic Aid, Am. J. M. Sc. **180**:336-356 (Sept.) 1930.

viz., that "if there is any band of nuclear tissue except a chromatin filament uniting the nuclear masses, these parts must not be considered as separate segments." This standard gives higher nonfilamented cell counts than those done with observance of less strict rules of differentiation, but the resulting figures are more accurate because of the sharper end point.

No attempt has been made to tabulate the number of cells showing cytoplasmic granules, vacuolation or other signs of toxic damage in these cases, since such a tabulation provides no special information not obtainable from the rest of the blood picture.

TABLE 1.—*White Blood Cell Pictures in First Four Cases of Burns*

Date, 1944	White Blood Cells, Number	Neutrophils, %	Myelocytes, %	Lymphocytes, %	Monocytes, %	Eosinophils, %	Basophils, %	Nonfilamented Neutrophils, %
Case 1, Second and Third Degree Burns of 85 per Cent of Body								
4/5	8,500	94.5	1.0	4.0	1.5	0	0	99
4/6	5,300	90.5	1.0	7.0	2.5	0	0	98
4/8	8,850	88.5	0	9.5	2.0	0	0	95
4/9	Died							
Case 2, Second and Third Degree Burns of 65 per Cent of Body								
4/ 6	10,400	88.0	1.0	8.0	3.0	1.0	0	81
4/ 7	14,800							
4/ 8	10,800	83.5	0.5	15.5	0.5	0.5	0	92
4/10	15,900							
4/17	24,900	74.5	0	24.0	0	0	1.5	95
4/18	Died							
Case 3, Second and Third Degree Burns of 35 per Cent of Body								
4/ 5	23,000	84.0	0	10.5	5.5	0	0	55
4/ 8	15,700	83.0	0	12.0	2.5	1.5	1.0	73
4/19	16,200	68.0	0	17.0	13.0	1.0	1.0	75
4/24	17,850	77.5	0	12.5	8.0	0.5	1.5	71
5/22	17,700	60.0	0	30.5	4.5	4.5	0.5	48
5/27	Discharged, to return for skin grafting							
Case 4, First and Second Degree Burns of 35 per Cent of Body								
4/ 5	17,300	75.0	0	17.5	7.5	0	0	57
4/10	10,600	63.0	0	19.0	9.0	9.0	0	58
4/19	10,100	61.5	0	26.5	9.5	2.5	0	47
4/29	Discharged							

THE DEGENERATIVE BLOOD PICTURE

In comparing the blood studies in the 4 cases of burns (table 1) it is evident that the total blood counts when first reported are not directly proportional to the severity of the injury. This might have been the case if counts had been made on April 3, 1944, the day of the burn, but by April 5 and 6 the counts in the cases of severer burns (cases 1 and 2) were lower, not higher, than those in the cases of milder burns (cases 3 and 4).

The presence of relatively low total leukocyte counts when much higher ones are expected, as evident in cases 1 and 2, has been generally assumed to be due either to an overstimulation of the leukopoietic mechanism such as to wear out productive ability or to a lack of resistance on the part of the patient. Neither of these explanations is the true one. These blood pictures demonstrate the presence of an often unrecognized factor in leukopoiesis, that of toxic inhibition. This toxic inhibition affects both the quantity and the quality of the white cells in the peripheral blood, resulting in the "degenerative" white blood cell picture.

The degenerative blood picture was first described by Schilling-Torgau [46] in 1911. He showed that there were two main types of shift of the neutrophilic white cells to the left and not just one type, as Arneth [47] had described in 1904. These two types are the regenerative and the degenerative. The regenerative type is found in ordinary pyogenic infections and other inflammatory conditions. It is characterized by leukocytosis, neutrophilia and, as the stimulation increases, more immature forms of neutrophils, such as juvenile forms, myelocytes and, eventually, myeloblasts. The shift to the left is seldom more than moderate (40 to 60 per cent of the neutrophils), even in the presence of myelocytes and myeloblasts.

The degenerative type of shift, on the other hand, is found, according to Schilling,[48] in a number of conditions, which include typhoid, malaria, tuberculosis and many virus diseases,

46. Schilling-Torgau, V.: Kritik der Arnethschen Lehre von der Verschiebung des leukozytären Blutbildes und Wertung ihren klinischen Anwendbarkeit, Folia haemat. **12**:130-177 (July 4) 1911.

47. Arneth, J.: Die neutrophilen Leukozyten bei Infektionskrankheiten, Deutsche med. Wchnschr. **30**: 54 and 92, 1904.

48. Schilling, V.: The Blood Picture and Its Clinical Significance, St. Louis, C. V. Mosby Company, 1929, pp. 86, 140, 151, 159, 180, 186 and 265.

such as influenza, measles, typhus and kala-azar. It is characterized by low rather than high total white cell counts and a differential count which is essentially normal except for a decided neutrophilic shift to the left. This degenerative shift is characteristically much more pronounced than the regenerative, often reaching to 90 per cent and over, and without the presence of young forms. Whereas the regenerative shift is a result of stimulation to leukopoiesis, the degenerative shift is due to toxic inhibition, representing, according to Schilling, "toxic-degenerative injuries of the bone marrow" affecting the peripheral cells.[48]

Recently the degenerative type of shift has been noted in several conditions not mentioned by Schilling, notably in diseases involving the gastrointestinal tract. These include paralytic abdominal distention,[49] "acute peritoneal irritation," [50] intestinal obstruction,[51] and abdominal distention from whatever cause.[52] In uncomplicated abdominal distention I found the degenerative blood picture occurring in essentially pure form; i. e., without regenerative admixtures.[52] From these studies it has been possible to separate the regenerative and degenerative blood pictures, and in table 2 are listed the distinguishing characteristics of each.

TABLE 2.—*Distinguishing Characteristics of the Degenerative and Regenerative Blood Pictures, Respectively*

Degenerative (Due to Toxic Inhibition)	Regenerative (Due to Inflammatory Stimulation)
Normal total count or leukopenia	Leukocytosis (may be a terminal drop)
Normal neutrophil count or neutropenia	Neutrophilia
Absence of juvenile forms and myelocytes	Tendency for juvenile forms and myelocytes to be present
Normal or relatively increased lymphocyte count	Relative decrease of lymphocytes
Normal or increased monocyte count	Decrease or absence of monocytes
Tendency for nonfilamented neutrophil count to be decidedly increased (65 to 95%)	Slight to moderate increase of nonfilamented neutrophils (30 to 60%)
Degenerative changes (toxic granules, vacuoles, pyknosis, etc.) in most of the cells	Absence of degenerative changes (except in very severe sepsis)

Schilling expressed the belief that certain blood pictures represent the antagonistic action between these two shifts, resulting in a mixed degenerative-regenerative type of shift. This mixed picture is found, for example, in influenza with pneumonia and in typhoid with peritonitis.[28] In those instances in which pronounced inhibition and stimulation are both present at the same time the total white cell count is usually somewhat elevated, though not as high as if no inhibition were present; the nonfilamented cell count may be higher (even over 95 per cent) than it ever goes in either type of shift alone; there is monocytopenia, disappearance of eosinophils and a tendency for myelocytes to be present, while the neutrophils and lymphocytes are variable. This mixed degenerative-regenerative blood picture has been illustrated and compared with the pure degenerative picture in a previous article [52] (table 5 of the article).

COMMENT ON THE FOUR REPORTED CASES OF BURNS

In the light of table 2, the blood pictures in the 4 cases of burns already reported may be more adequately interpreted. In case 1, the unexpectedly low counts and the extremely high percentages of nonfilamented cells are now seen to be degenerative characteristics which signify toxic inhibition. The neutrophilia, presence of myelocytes, monocytopenia and absence of eosinophils are all regenerative changes, indicating marrow stimulation. Thus a pronounced toxemia together with a high degree of inflammatory stimulation is shown by the blood picture. This stimulation was explained at necropsy by the finding of bronchitis and bronchopneumonia.

In case 2 the toxic inhibition is less than in case 1, as shown by the higher total count and lower percentage of nonfilamented cells on April 6. It is still considerable, however, and affects the picture throughout. The last counts (April 10 and 17) show evidence of increasing stimulation in the higher total counts and disappearance of monocytes and eosinophils, the source of the stimulation being presumably the gangrene of the foot and the pneumonia.

In cases 3 and 4 the regenerative effect is shown by the pronounced leukocytosis in each on April 5, the second day after the burn. In case 3, there is evidence of some toxicity in the increased percentage of nonfilamented cells shown in the next three counts. In case 4 there appears to be little toxicity.

49. Van Duyn, J., II: Leukocyte Exhaustion Following Surgical Procedures, Arch. Surg. **37**:302-310 (Aug.) 1938.

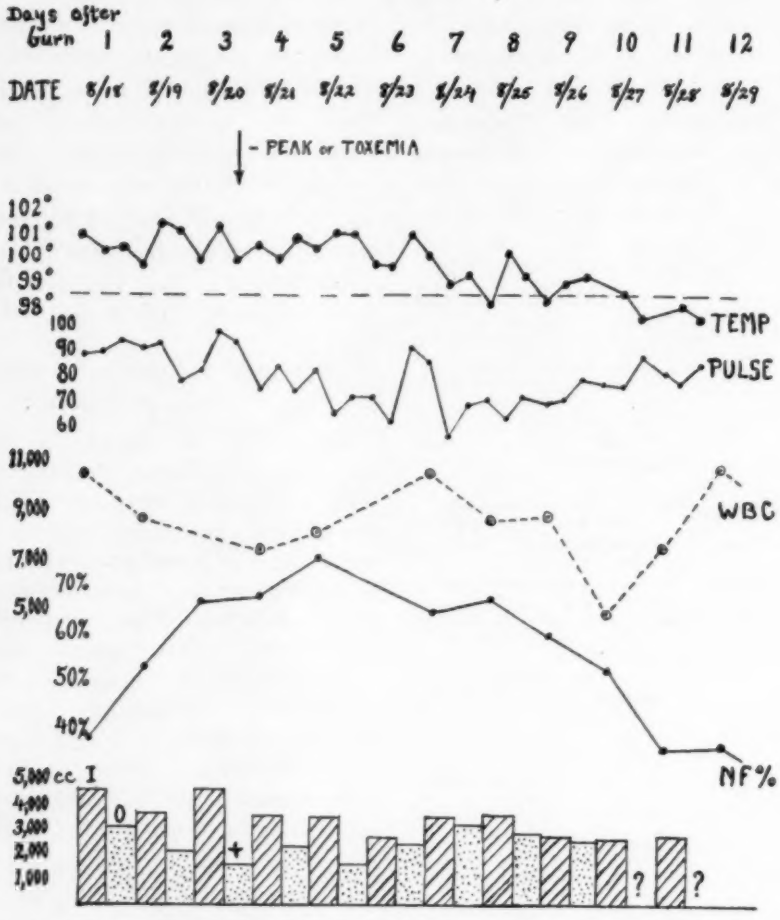
50. Kaufman, R. E., and Vom Saal, F.: Leukocyte Changes in Acute Peritoneal Irritation, J. Lab. & Clin. Med. **26**:468-476 (Dec.) 1940.

51. Harris, F. I., and Feldheym, J. S.: Leukocyte Exhaustion in Intestinal Obstruction, Am. J. Surg. **54**:417-423 (Nov.) 1941.

52. Van Duyn, J., II.: Role of Abdominal Distention in Leukocyte Exhaustion, Arch. Surg. **44**:339-352 (Feb.) 1942.

COMMENTS ON THREE MISCELLANEOUS CASES

Sixteen per Cent Second Degree Burns.—Although it is not so important to attempt an estimate of the degree of toxemia in burns of small extent, it may be easier to do so because of the fewer complications; and it is interesting to correlate the effect of this pure toxemia on the white blood cells with that on the rest of the clinical picture.



Laboratory data for L. D. (case 5), admitted to the hospital with 16 per cent of his body covered with second degree burns. Details are given in table 3.

In the chart are the laboratory data in a case (case 5) of second degree burns of the face, neck, chest and hands covering an estimated 16 per cent of the body surface.

CASE 5.—L. D., a 21 year old man, was admitted to the Syracuse Memorial Hospital on the evening of Aug. 17, 1944 two hours after being burned in a gasoline explosion.

The treatment consisted of use of cod liver oil ointment dressings locally and sulfadiazine orally. No plasma was administered. There were no complications. No skin grafts were required, although for two areas of the hands it was thought at one time they might be necessary. The hematocrit determination twelve hours after the patient's admission was 53 volumes per cent.

From the chart the white cell count and percentages of nonfilamented cells show clearly the onset of toxemia. It is interesting to compare these with the temperature, pulse rate and fluid intake and output. On the morning of August 21 (three and one-half days after the burn) the total white cell count reached its toxic low point of 7,300 per cubic millimeter and the pulse rate began to fall. On August 22 (four and one-half days after the burn) the count of nonfilamented cells reached its peak of 75 per cent. On August 23 (five and one-half days after the burn) the relative urinary output returned to normal or above and the temperature began to drop. By the seventh day all values except the nonfilamented cell count had returned to normal.

Since there is probably some lag between the absorption of toxins into the body and their

effect on the peripheral blood picture, it may be assumed that toxicity reached its peak in this case about seventy-two hours after the burn and had largely subsided by the morning of the seventh day. The nonfilamented cell count was valuable as an early indicator of toxemia, climbing significantly between the first and second

TABLE 3.—*Details of Blood Counts in a Case of 16 per Cent Second Degree Burns (Case 5)*

Date, 1944	White Blood Cells, Number	Neutrophils, %	Lymphocytes, %	Monocytes, %	Eosinophils, %	Basophils, %	Nonfilamented Neutrophils, %
8/18	10,400	87	2	11	0	0	36
8/19	8,800	72	10	14	3	1	54
8/20	.....	86	4	10	0	0	66
8/21	7,300	82	6	8	4	0	67
8/22	8,000	59	15	19	6	1	75
8/23							
8/24	10,200	64	23	9	4	0	63
8/25	8,500	58	30	9	2	1	66
8/26	8,600	66	19	11	4	0	58
8/27	4,600	65	24	10	1	0	51
8/28	7,400	74	14	9	1	2	35
8/29	10,500	67	20	10	3	0	36
8/30	8,900	65	21	9	5	0	29

given the white blood cell picture and the figures for fluid intake and output in a case of a burn which was much more extensive than that of L. D. but far less toxic.

CASE 6.—N. W., a 45 year old man was admitted to Crouse-Irving Hospital on Dec. 3, 1944 and discharged as recovered on Jan. 2, 1945. He was burned when a can of kerosene exploded, the burn involving parts of the trunk and both extremities. According to the Berkow table, the second degree area covered 30 per cent of the body surface, and the first degree area 10 per cent. The hematocrit reading was 49 volumes per cent on the morning after admission. Treatment consisted of petrolatum gauze pressure dressings locally, 1,000 cc. of plasma intravenously and sulfamerazine orally. Recovery was rapid, the patient being up and about on the seventh day after the burn. There were no complications; no third degree areas appeared, and no areas required grafting.

This case illustrates the value of the white cell picture in demonstrating the mildness of a toxemia. As the table shows, toxemia was so slight that even with the help of the regenerative stimulation always present the percentage of

TABLE 4.—*Blood Counts and Fluid Intake and Output in a Case of 40 per Cent First and Second Degree Burns (Case 6)*

Date, 1944	White Blood Cells, Number	Neutrophils, %	Lymphocytes, %	Monocytes, %	Eosinophils, %	Basophils, %	Nonfilamented Neutrophils, %	Intake, Cc.	Output, Cc.
12/ 4.....	20,000	74	19	6	1	0	38	3,860	1,230
12/ 5.....	.....	..	..	..	..	..	..	2,500	1,080
12/ 6.....	16,850	70	20	10	1	1	43	1,980	780
12/ 7.....	13,700	85	11	3	0	1	42	3,150	1,800
12/ 8.....	.....	..	..	..	..	..	..	2,040	2,050
12/ 9.....	.....	..	..	..	..	..	..	2,280	2,340
12/10.....	.....	..	..	..	..	..	..	2,090	2,240
12/11.....	11,700	65	21	12	2	0	26	1,720	990

days. It fell to normal so slowly, however, that it was still definitely elevated as late as the tenth day after the burn, three days after all other signs of toxemia had disappeared.

The sharp drop in the white cell count on August 27 and the rise on August 29 are not mistakes. Such oscillations are frequent during the readjustment phase of a hemopoietic strain. In addition to the chart, a table (table 3) showing the more detailed differential findings is presented for case 5.

It is also interesting to compare the first count in table 3, August 18, when the stimulation of the burn trauma predominated and before the onset of toxemia, with those on August 21 and 22, when the toxic effect was at its peak. The former shows purely regenerative characteristics, except for the slight monocytosis, and the latter purely degenerative, except for the slight neutrophilia on August 21.

First and Second Degree Burns Covering 40 per Cent of the Body Surface.—In table 4 are nonfilamented cells reached a peak of only 43 per cent (on December 6). It was insufficient to depress the total white cell count, which during the toxic phase showed a level of 13,700 per cubic millimeter. The only evidence of toxemia, besides the nonfilamented cell count, was in the lowered relative renal output of the first four days.

Intestinal Obstruction.—Patients will die after the too sudden release of an intestinal obstruction while exhibiting the same high fever, collapse, semicoma and other symptoms of toxic shock as in burns. The presence of toxemia is also similarly reflected in the blood picture,[53] as illustrated by the following case of uncomplicated intestinal obstruction.

CASE 7.—H. F., a 29 year old man, was admitted to the University Hospital of the Good Shepherd Aug. 26, 1944, with symptoms of low intestinal obstruction of five days' duration. Intestinal intubation failed to relieve an increasingly pronounced abdominal distention, and on September 1 the abdomen was opened. A kink at

53. Harris and Feldheym.[51] Van Duyn.[52]

the splenic flexure of the colon was the only pathologic condition found, and a cecostomy was performed. After the cecostomy, the distention subsided rapidly and the patient improved steadily. No explanation for the obstruction was found either at operation or on subsequent examination with a barium sulfate enema, though a history of congenital syphilis may be significant.

Table 5 gives the white cell and differential counts in the case of H. F. The blood picture on September 1, taken at the height of the distention and just before the cecostomy, shows the greatest evidence of toxicity; i. e., the largest number of degenerative characteristics. On this date the total count was at a leukopenic level (4,000 cells per cubic millimeter) and the percentage of nonfilamented cells was extremely high (96 per cent). This blood picture shows greater toxicity than the corresponding count in the case of uncomplicated burn (L. D.) (see table 3, August 22), but their essential similarities are obvious.

TABLE 5.—*Laboratory Data in a Case of Intestinal Obstruction (Case 7)*

Date, 1944	White Blood Cells, Number	Neutrophils, %	Lymphocytes, %	Monocytes, %	Eosinophils, %	Basophils, %	Nonfilamented Neutrophils, %	Comment
8/20	.....	....	....	...	...	...	..	Admitted
8/26	15,000	....	....	...	...	...	..	First nausea
8/28	14,700	66.0	20.0	7.5	4.5	2.0	33	Hemoglobin, 107 per cent; red blood cells, 5,660,000
8/31	.....	....	....	...	...	...	..	Marked distention
9/ 1	4,000	58.0	33.5	8.0	0	0.5	96	Nonprotein nitrogen, 38 mg. per 100 cc.; cecostomy
9/ 2	11,700	53.5	22.5	20.5	2.5	1.0	93	Hemoglobin, 93 per cent; red blood cells, 4,530,000; plasma proteins, 6.5 Gm. per 100 cc.; alkaline phosphatase, 4 B. U.
9/ 3	.....	....	....	...	...	...	..	Cephalin flocculation 2 plus
9/ 5	14,900	66.0	22.5	8.0	3.5	0	67	
9/13	14,200	63.0	30.0	2.5	4.0	0.5	26	Barium sulfate enema
9/29	.....	....	....	...	...	...	..	Discharged recovered

Thus, there is hematologic evidence of the presence of a similar toxic substance in intestinal obstruction and burns.

THE SEVERAL TOXINS IN BURNS

It should be remembered that there are a number of different toxic substances in burns, all affecting both the clinical and the laboratory picture. The first to appear is the toxin associated with the tissue damage of immediate injury, the "traumatic" toxin mentioned by Harkins [28]; next, if it occurs, is the true burn toxin, and, finally, there may be bacterial, necrotic tissue and other toxins. By true burn toxin I have meant only that apparently nonspecific substance which has an inhibitory effect on the leukopoietic organs resulting in the degenerative white cell picture. The other toxins are apparently all stimulatory, and cause only regenerative changes. The substance responsible for the leukopoietic stimulation in these stimulatory toxins is probably identical with Menkin's "leukotaxine." [54]

PROGNOSIS AND TREATMENT OF BURN TOXEMIA ACCORDING TO THE BLOOD PICTURE

If a patient with a severe burn survives the first two or three days, he is then past the dangers of primary and secondary shock and within the period of true burn toxemia. By this time there should be marked degenerative changes in the white cell picture if toxemia is to be a significant factor. According to the blood pictures in my cases, toxemia may be evident by at most the second day after the burn. It probably reaches its height somewhere between the third and sixth days,[12] but deaths occurring up to ten days afterward may still be considered due to toxemia.[23]

From the first to at least the third or fourth day following a burn, the white blood cell picture must be closely watched if the onset of toxemia is to be predicted. In the cases of severest toxemia the degenerative changes come on rapidly and are extreme. An example of such a picture is apparently that in McIver's case,[42] in which a white cell count of 1,000 per cubic millimeter (the only count reported) was present on the third day after the burn. The 2 cases mentioned by McSwain [6] in which the patients died of "agranulocytosis" may also have been actually of this type. Such cases probably belong in the group of inevitably fatal cases and are perhaps comparable to Pfeiffer's type 1 observations.[40]

54. Menkin, V.: XV. Studies on Inflammation: Concerning the Mechanism of Cell Migration, J. Exper. Med. **67**:145-158 (Jan.) 1938.

If toxemia is not extreme but still severe, the prognosis may depend on whether or not regenerative changes are present. If the changes are mixed regenerative-degenerative and of marked degree, it indicates that some complicating infection has developed, such as bronchitis or pneumonia. In such cases, the prognosis is still poor, but it is probably in just such cases that the best hope lies of further lowering the mortality rate of burns. This picture is exemplified best by the counts in cases 1 and 2 (table 1). It corresponds closely with the blood picture described by Schreiner and Pucsko [41] as indicating a bad prognosis and that described by Fasal [48] as demanding immediate and repeated blood transfusions.

In cases with severe regenerative pictures without degenerative admixtures the prognosis is probably better than in those showing evidence of severe toxemia too. It should still be guarded, however, particularly if the evidence of irritation is intense, indicating the presence of extensive necrosis or some complicating infection or both.

Unfortunately, there is still no specific antidote for the toxemia of burns any more than for that of intestinal obstruction. Transfusions of whole blood, however, do have considerable value and have apparently been somewhat neglected since hemoconcentration and the use of plasma have held the spotlight. It is possible that just as plasma was found to be required in much greater quantities for secondary shock than was originally supposed necessary, whole blood will prove effective in many cases of toxemia if given in much larger amounts than generally done at present. The blood transfusions should not be started, of course, until hemoconcentration is corrected and need not be given at all during this phase unless toxicity is predicted. The amount of blood required can be regulated by the "degeneracy" of the blood picture, just as the amounts of plasma are regulated by hematocrit determinations.

The idea of giving large blood transfusions to combat burn toxemia is not new. Harkins' text includes a list of authors who have advocated this treatment through many years, and even shows that the subject of exsanguination transfusions is not closed. Now that toxemia is predictable by means of the degenerative blood picture, blood transfusions can be started earlier, before toxemia has become irreversible, and given in more adequate amounts without fear of wasting them on nontoxic patients.

There is one other therapeutic agent besides whole blood that deserves a trial in treatment of burn toxemia, and that is liver extract. Injections of crude liver extract have already proved their value for patients showing the degenerative blood picture,[55] and also, interestingly, for experimental burns.[56] It may be that liver extract acts through its beneficial effect on hepatic function in general and its detoxifying ability in particular.[57] Doses of 10 cc. intramuscularly during each twenty-four hours of the toxic phase should be adequate to produce a maximal effect.

CONCLUSIONS

There is an inhibitory effect on the leukopoietic system frequently demonstrable in burns which is distinct from the ordinary stimulatory effect produced by traumatized, infected or necrotic tissue.

This inhibitory effect is characterized by "degenerative" changes in the white blood cells which can be differentiated from the "regenerative" changes resulting from ordinary stimulation.

Whereas the regenerative blood picture is due to inflammatory stimulation, the degenerative blood picture is a result of toxic inhibition.

There is, therefore, a true toxemia in burns, distinct from trauma and hemoconcentration, on the one hand, and sepsis, on the other, and due to the absorption of some toxic substance from the burn area.

By observing the degree of degenerative changes in the white cells the presence of toxemia can be recognized and its severity estimated. This is more difficult when the picture is a mixed degenerative-regenerative one since inhibition and stimulation are both acting together.

The degenerative white blood cell picture is found in many conditions besides burns—for

55. Powers, J. H., and Murphy, W. P.: Leukocytosis Following the Intramuscular Injection of Liver Extract, J. Clin. Investigation **12**:713-721 (July) 1933. Meyer, O. O.; Middleton, W. S., and Thewlis, E. M.: Leukocytosis After Parenteral Injection of Liver Extract, Am. J. M. Sc. **188**:49-60 (July) 1934. Powers, J. H.: Further Observations on the Leukocytic Response Induced by the Intramuscular Injection of Liver Extract, J. Clin. Investigation **14**:649-657 (Sept.) 1935. Van Duyn.[49]

56. Prinzmetal, M.; Margoles, C., and Feigen, G.: A Principle from Liver Effective Against Shock Due to Burns, J. A. M. A. **122**:720-723 (Jan. 10) 1943.

57. Büttner, H. E.: Experimentelle und klinische Untersuchungen über die Wirkung einspritzbarer Leberextrakte, Fortschr. d. Therap. **11**:257-264 (May) 1935. Astrachan, G. D.: Crude Liver Extract as Aid to Arseno- and Heavy-Metal Therapy, New York State J. Med. **44**:992-1003 (May 1) 1944.

example, intestinal obstruction—showing that the inhibiting toxin is not specific.

During the toxic phase of burns the prognosis may depend on the degree of degeneracy of the white blood cell picture. If the picture is mixed degenerative-regenerative, the prognosis is worse than if a similar degree of degenerative change is present alone.

The giving of large whole blood transfusions, begun immediately after the period of hemoconcentration and repeated as indicated by the appearance and disappearance of degenerative changes in the white cells, is probably the best general measure available to combat the toxemia of burns.

It is suggested that the injection of crude liver extract be combined with the giving of blood transfusions during the toxemic phase because of its known beneficial effect on the degenerative white blood cell picture.

Dr. Leonard M. Aquilino gave me permission to study his cases, Lieut. Robert Seidenberg called a case to my attention, and Dr. Warren R. Sisson supplied some of the data and blood smears. The staff of the library of Syracuse University College of Medicine cooperated in the study.

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CONGENITAL MALFORMATIONS OF THE ANUS AND RECTUM

A CLINICAL STUDY

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Congenital malformations of the anus and rectum are rather uncommon and are said to occur about once in every five thousand newly born babies. According to Allen,[1] David[2] and Gant,[3] these deformities occur about once in every ten thousand births. A survey of the records at St. Mary's Hospital, which was opened in 1924, showed no anomalies of the anus and rectum until 1931, and from 1931 to the present time (1944) a total of 15 cases have been collected. During this period of twenty years, a total of 118,809 admissions were recorded, with 0.0013 per cent of the total admissions caused by the anomalies. Ladd and Gross,[4] working at Children's Hospital in Boston, collected a total of 214 cases over a period of thirty-one years. Crowell and Dulin[5] collected 28 cases over a period of twelve years at the University of Iowa Hospitals.

Anomalies of the anus and rectum are classified by Ladd and Gross[6] into four types, as follows:

1. Incomplete rupture of the anal membrane or stenosis at a point 1 to 4 cm. above the anus.

2. Imperforate anus with the obstruction due only to a persistent membrane.

3. Imperforate anus with the rectal pouch separated from the anal membrane. The rectal pouch may end blindly either in or above the pelvis.

4. Normal anus and anal pouch with the rectal pouch ending blindly. There may be either a membranous obstruction between or a separation of the anal and rectal pouches. When the pouches are separated, a cord of tissue without a lumen occasionally may connect them.

Of the 15 patients, 5, or 33.3 per cent, had anomalies of type 1, and 10, or 66.6 per cent, of type 3. There were no anomalies of type 2 or type 4 represented in this series. The sex distribution showed 4 males and 1 female with anomalies of type 1, and 8 males and 2 females with anomalies of type 3 (table 1).

TABLE 1.—*Distribution of the Four Types of Anomalies*

Type	Males	Females	Total
1	4	1	5
2	0	0	0
3	8	2	10
4	0	0	0
Totals	12	3	15

Five patients, or 33.3 per cent, had fistulas connecting the rectum with the genitourinary tract or perineum. Of these, 2 were male and 3 were female. Two (16.6 per cent) of the males and 3 (100 per cent) of the females had associated fistulas. One of these fistulas occurred in association with a type 1 anomaly and 4 in association with a type 3 anomaly. This is in agreement with the statistics reported by Ladd and Gross.[6]

TABLE 2.—*Distribution of Fistulas in Males and Females*

Type of Fistula	Males	Females	Total
Rectovesical	1	0	1
Rectourethral	1	0	1
Rectovaginal	0	2	2
Rectum–fossa navicularis	0	1	1
Totals	2	3	5

Two types of fistula were encountered in the males, rectovesical and rectourethral. No rectoperineal fistulas were found. Of the females, 2 had rectovaginal fistulas and 1 had a rectum–fossa navicularis fistula. No rectovesical,

From the Department of Surgery, St. Louis University School of Medicine.

1. Allen, V. K.: Malformations of the Anus and Rectum, Tr. Am. Proct. Soc. **33**:148-153, 1932.

2. David, V. C.: Embryology and Malformations of the Rectum, in Nelson's Loose Leaf Living Surgery, New York, Thos. Nelson & Sons, 1937, vol. 5, pp. 161-164.

3. Gant, S. G.: Diseases of Anus, Rectum and Colon, Philadelphia, W. B. Saunders Company, 1923.

4. Ladd, W. E., and Gross, R. E.: Congenital Malformations of Anus and Rectum, Am. J. Surg. **23**: 167-183, 1934.

5. Crowell, E. A., and Dulin, J. W.: Congenital Anomalies of the Anus and Rectum, Surgery **7**:529-539, 1940.

6. Ladd, W. E., and Gross, R. E.: Abdominal Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1941; footnote 4.

rectoperineal or combined rectovaginal and rectovesical fistulas were encountered.

Five patients, or 33.3 per cent, had other associated congenital anomalies. These included absence of the gallbladder, horseshoe kidney, hydroureter and hydronephrosis, patent ductus arteriosus, anomaly of the ureters, hypospadias and unusually large foramen ovale and mongolian idiocy. The anomaly of the ureters was rather unusual. The ureters fused into one which emptied into the bladder on the right side, and the colon entered the bladder on the left side. In no instance was death directly attributable to any of the associated anomalies. All these associated congenital anomalies occurred in males with type 3 malformations.

SYMPTOMS

The symptoms presented by the patients varied with the type of anorectal anomaly and the presence or absence of associated fistulas.

Type 1.—The age range of patients with type 1 anomalies was from 6 weeks to 22 months, with an average age of 9 9/10 months. These patients never manifested symptoms of intestinal obstruction, although 1 of them became moderately distended, and were therefore seen later than those with other types of anomalies. The usual complaints were small, thin, frequent bowel movements associated with pain and crying. One child had liquid bowel movements and was found to have a fecal impaction. One mother noticed a very small anal orifice. Another noticed some bleeding from the rectum for three days after the child had been straining and having cramps with each attempted bowel movement for four weeks.

Type 2.—Although no anomalies of type 2 were encountered in my patients, the symptoms expected from the pathologic observations and reported in the literature will be mentioned for the sake of completeness. The infants were usually seen within the first three days of life. The mother or the physician readily noticed the imperforate anus and the absence of meconium on the diapers. During the first thirty-six hours the absence of bowel movements was usually the only symptom, but after that period symptoms of intestinal obstruction appeared.

Type 3.—Of the 10 patients with type 3 anomalies, 7 were seen within the first two days of life, 2 were seen three and six weeks after birth respectively and 1 was seen ten months after birth. Four patients in the group had associated fistulas. The patient seen ten months after birth had an adequate fistulous opening in the vagina through which the bowels moved, and she showed no evidence of intestinal obstruction. Three other patients had fistulas and were seen within the first two days of life. Two of these had adequate fistulous openings to permit the passage of fecal material and gas and therefore did not present symptoms of intestinal obstruction. The other one had a rectourethral fistula whose opening was evidently inadequate, because obstructive symptoms developed in a period of twenty-four hours.

There were 6 patients without associated fistulas in this group. Three of them presented definite symptoms of intestinal obstruction; 1 had a proctoplasty within twenty-four hours, before obstructive symptoms could develop, and 2 were seen three weeks and six weeks respectively after birth with double-barreled colostomies in the left lower abdominal quadrant.

Type 4.—Patients with type 4 anomalies are usually seen later than those with other types because the normal-appearing anus favors overlooking the possibility of a congenital anomaly. As a result, the intestinal obstruction which develops is of longer standing and the patients are more "toxic" and therefore poorer surgical risks.

PHYSICAL OBSERVATIONS

The observations on physical examination varied with the type of anorectal anomaly present and the age of the patient. The age is particularly important in the case of patients having a malformation which produces a complete occlusion of the anus or rectum. The patients acquire the signs characteristic of intestinal obstruction, namely abdominal distention, tympanites, intestinal patterning, increased borborygmi, vomiting and dehydration after twenty-four hours. Examination of the perineum will give one the information required to classify the type of anomaly and determine the presence or absence of a fistulous opening by observing the passage of fecal material or flatus from an abnormal site.

Type 1.—The anal opening ranged from 1 to 5 mm. in diameter, with an average diameter of 2 mm. One patient had an anal canal approximately 1 inch (2.5 cm.) long which admitted a fine probe. None of these patients had an obstruction higher in the anal canal. The colon in each patient was dilated and filled with fecal material. The external opening of a rectum–fossa navicularis fistula was observed in 1 patient.

Type 2.—In patients with type 2 anomalies the anal membrane is imperforate and discolored by the meconium. When the children strain or cry the impact of the meconium makes the membrane bulge.

Type 3.—Patients with this type of anomalies presented a variety of physical characteristics. The anal area had a dimple in 5 patients, and in the other 5 patients there was no ridge or dimple in the median raphe to denote the normal anal site. In 1 patient only was the blindly ending rectal pouch low enough to impart an impulse to the anal region on crying. In 2 male patients, meconium and gas were observed passing from the penile urethra. Two female patients had openings in the vagina through which fecal material was expelled. Three of the 4 patients had either a catheter or a probe inserted into the fistulous tract in an attempt to determine the position of the rectal pouch.

Type 4.—The anus is normal in appearance, but digital examination of the rectum reveals a complete obstruction within reach of the examining finger. The external sphincter is present and contracts on stimulation.

ROENTGENOGRAPHIC OBSERVATIONS

Roentgenographic studies of the intestinal tract were made for 9 of the 15 patients. Three in the group with type 1 malformation and 6 in the group with type 3 malformations.

The roentgenograms of the infants with type 1 anomalies revealed pronounced dilatation of the colon due to the gas and fecal material dammed back behind the constricted anus. These roentgenograms were merely flat plates of the abdomen for 2 patients and a flat plate made after the injection of a barium sulfate mixture in the form of an enema for the third patient.

Three different types of roentgenographic studies were utilized for the patients with type 3 anomalies. Of the 6 patients in this group, 1 had a series of roentgenograms of the gastrointestinal tract, 2 had barium sulfate mixture injected into the distal loop of the colostomy and 2 had flat plates of the abdomen in the upright and the inverted positions followed by a colostomy and subsequent barium studies of the distal loop of the colostomy. The purpose of these studies was to ascertain the position of the blind end of the sigmoid colon or rectum. Wangensteen and Rice[7] advocated the procedure of taking flat plates of the abdomen and pelvis with the child inverted and an opaque marker in the anal region. By this method the gas in the intestines will rise and fill the rectal pouch, giving an indication of its position with relation to the anal membrane. This procedure is of value only after twenty-four hours, when a sufficient amount of gas has formed to fill the intestines. Giving a barium meal for a series of gastrointestinal roentgenograms is contraindicated for these patients, just as for any other patient with intestinal obstruction.

7. Wangensteen, O. H., and Rice, C. O.: Imperforate Anus—A Method of Determining the Surgical Approach, Ann. Surg. **92**:77-81, 1930.

TREATMENT

Treatment of congenital malformations of the anus and rectum also varies with the type of malformation present.

Type 1.—Of the 5 patients in this group, 3 were treated simply by rectal dilations. Of the other 2 patients, the one with a pinpoint opening had a simple proctoplasty which widened the anus so that the thumb could easily be inserted. The other patient had a rectum–fossa navicularis fistula with an orifice measuring 2 mm. in diameter. This patient was first seen when 19 months old and was treated by dilation of the orifice of the fistula and removal of a fecal impaction. This child was seen again when 2½ years of age, at which time a plastic operation was performed and the fistulous tract mobilized and placed in the proper position, with the opening at the normal anal site.

Type 2.—The accepted form of treatment for type 2 anomalies is simple cruciate incision of the anal membrane followed by anal dilations.

Type 3.—Treatment for persons with this type of anomalies was dependent on the presence or absence of obstructive symptoms.

Five patients had no obstructive symptoms and therefore had primary perineal operations. Four of these patients were operated on within the first two days of life and 1 after ten months, when she was first seen, with a rectovaginal fistula through which the bowels moved. An incision was made in the midline, and the dissection carried upward, separating the transversus perinei and levator ani muscles. In 2 patients the rectum was penetrated and the peritoneum was opened accidentally. The wounds were packed, and the patients died twenty-four and thirty-six hours postoperatively with peritonitis. In the other 3 patients the rectum was mobilized and brought down to the skin incision, opened and then sutured to the skin edges. On 2

of these patients rectal dilation was done postoperatively starting ten days after operation. In 1 of these 2 patients an anal stricture developed; it was treated by a plastic operation and further dilation.

Five patients had obstructive symptoms and had sigmoid colostomies performed. One of these patients was first seen when 3 weeks old and another when 6 weeks old. Both had a sigmoid colostomy performed elsewhere when they were 2 days old. These patients had a perineal plastic operation as previously described. One died on the ninth postoperative day with peritonitis due to kinking of a loop of small intestine beneath the colostomy loop and resultant obstruction. The other died on the fifth postoperative day with a clinical picture of peritonitis. Two of these patients had primary sigmoid colostomies. One also had several inches of small intestine resected, followed by an antiperistaltic side to side anastomosis. This was done because of trauma to the small intestine and its mesentery. This patient died from shock on the day after operation. The other patient had a poorly functioning colostomy, and part of it became gangrenous and required excision. This patient had a rectourethral fistula, and pyelonephritis, which was fatal, developed. One patient had a colostomy performed when 1 day old and then had a perineal plastic operation (as described previously) approximately three months later. This patient subsequently died with a lobular pneumonia, urinary suppression and tubular nephrosis seven months after birth. At autopsy this patient also had a horseshoe kidney and absence of the gallbladder.

Type 4.—The accepted form of treatment for this type of anomalies is performance of a colostomy for relief of the intestinal obstruction, especially when the patient is in poor condition. The perineal rectal repair is then done later at a second operation. Usually the rectal pouch is high up in the pelvis and the perineal approach is difficult, requiring extensive perineal exploration. With the two sacs usually separated from each other, the lumens are usually joined by an oblique anastomosis.

The choice of anesthesia for infants is limited to use of either local anesthesia or drop ether. Drop ether was used in all the cases except 1 in which local anesthesia was used.

There was no mortality in patients with type 1 anomalies during their stay in the hospital. One patient died of pneumonia four months after leaving the hospital. Until that time he was well and had good control of his bowels. The others are alive and well, with a normal-functioning anus, six months to six years after the operation. In the group with type 3 anomalies 7 of the 10 patients died during their stay in the hospital, a mortality of 70 per cent. Of the 3 patients who left the hospital alive, 2 are living and well four and five years after operation. One of the patients will have another operation to close a persistent rectovaginal fistula.

TABLE 3.—*Mortality in This Study*

Type of Anomaly	Males	Females	Total
1	0	0	0
2	0	0	0
3	7	0	7
4	0	0	0
Totals	7	0	7

At the present time, however, she has good control of her bowels, which move regularly, does not soil her clothes and does not pass feces from the vagina. A follow-up on the third patient that left the hospital alive was impossible, since both the physician and the surgeon who treated her are in the armed forces and the parents cannot be contacted. This patient was a mongolian idiot, and this condition in itself has a high mortality. It is probable that this patient is dead, since almost four years have elapsed since its birth. The average mortality for this series of patients was 46.6 per cent (tables 3 and 4).

TABLE 4.—*Mortality Reported in the Literature and in This Study*

	Ladd and Gross		Crowell and Dulin		St. Mary's	
Type of Anomaly	Mortality, %	No. of Patients	Mortality, %	No. of Patients	Mortality, %	No. of Patients
1	9.5	21	0	3	0	5
2	16.6	6	4.3	6	No cases	
3	24.8	117	13.0	16	70	10
4	61.4	18	0	3	No cases	

Ladd and Gross [4] reported a mortality of 9.5 per cent, 16.6 per cent, 24.8 per cent and 61.4 per cent in cases of type 1, 2, 3 and 4 anomalies respectively, the average mortality for the entire series being 28 per cent. Crowell and Dulin [5] reported a mortality of 17 per cent for 23 of the 28 patients treated surgically, 4.3 per cent and 13 per cent in cases of type 2 and type 3 anomalies respectively (table 4).

SUMMARY

Fifteen patients with congenital malformation of the anus and rectum were studied. In this group of patients only two of the four types of anomalies were represented. The treatment for the type 1 anomalies was satisfactory, but that for the type 3 anomalies was unsatisfactory, judging from the mortality in this group. Basically, the treatment was satisfactory in performing colostomies for patients with symptoms of intestinal obstruction and in attempting primary perineal plastic operations for those patients free of obstructive symptoms. Two patients who had primary perineal operations died from peritonitis as a result of perforation of the rectum and contamination of the peritoneal cavity. These were technical accidents. The 5 patients presenting obstructive symptoms and for whom colostomies were performed died. One patient with a type 3 anomaly was given a barium sulfate meal as a diagnostic aid. This is contraindicated as for any other patient with intestinal obstruction.

ACUTE APPENDICITIS IN CHILDHOOD

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In 1939 Hudson and Chamberlain [1] published statistics on 848 cases of appendicitis in patients treated at The Children's Hospital, Boston, during the ten year period preceding Jan. 1, 1939. Since that time there has been a significant decrease in the mortality of appendicitis in this clinic, and it is the purpose of the present study to evaluate the factors which are responsible for more successful therapy and to present an analysis of several aspects of the disease in childhood.

INCIDENCE

During the five and a half year period from Jan. 1, 1939 to June 30, 1944, there have been 506 children with acute appendicitis treated in this hospital. The various types encountered are listed in table 1.

TABLE 1.—*Types of Appendicitis in 506 Children*

Acute, unruptured without fluid	162
Acute, unruptured with fluid	110
Acute, ruptured with local peritonitis	96
Acute, ruptured with general peritonitis	90
Acute, ruptured with abscess	48

We have included in this series only proved cases of acute appendicitis with and without complication and have purposefully omitted all cases of appendectomy performed for other reasons.

Tabulation of sex incidence in this group of 506 cases reveals that 59 per cent of the patients were male and 41 per cent were female. This is in accord with the slightly greater predominance of males in most series of cases of adult patients.

Examination of the relation between type of appendicitis and age of our patients serves to support the view that perforation of the acutely inflamed appendix tends to occur earlier in the course of the disease in babies and young children than in persons in later life (table 2).

This is without question the age group in which early diagnosis is most difficult. It is interesting to note that in children under 6 years of age perforation was encountered more than twice as often as was simple acute appendicitis without rupture. All our patients under 2 years of age had perforated appendixes, with the single exception of an 11 month old boy who was operated on within twenty-four hours after the onset of symptoms.

There was no striking seasonal variation in the incidence of appendicitis in this five and a half year period.

TABLE 2.—*Relation Between Type of Appendicitis and Age*

Acute Unruptured		Acute Ruptured	
Age in Years	Number of Cases	Age in Years	Number of Cases
0- 1	1	0- 1	0
1- 2	0	1- 2	22
2- 4	15	2- 4	59
4- 6	37	4- 6	42
6-12	195	6-12	109
12-16	24	12-16	2

ETIOLOGIC ASPECTS

It has long been felt that infections of the upper respiratory tract bear a definite relation to appendicitis and that the latter has its greatest incidence during the so-called "respiratory seasons." However, in our series infections of the respiratory tract were present immediately prior to hospitalization for appendicitis in only 16 per cent of cases, and this combination bore no relationship to a particular season of the year. According to Hudson and Chamberlain,[1] this figure represents the expected incidence of infections of the respiratory tract in the average child population.

The only acute illnesses other than infections of the respiratory tract preceding appendicitis in

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1. Hudson, H. W., Jr., and Chamberlain, J. W.: J. Pediat. **15**:408, 1939.

this group were gastroenteritis (10 cases), rheumatic fever (4 cases), bronchopneumonia (3 cases) and individual instances of mumps, whooping cough, bacillary dysentery and rheumatoid arthritis. The extremely low incidence of such diseases would seem to preclude any causal connection with appendicitis. A history of previous attacks of unexplained abdominal pain was elicited in 82 cases, an incidence of 16 per cent. While some of these episodes may have been acute appendicitis, our data are insufficient for definite conclusions. Pathologic examination of the appendix in these cases revealed old scarring in addition to the acute process in only a few instances. By and large, in this series acute appendicitis developed rather suddenly in children who had been previously well.

Wangensteen and colleagues [2] have recently produced the pathologic and clinical features of acute appendicitis experimentally in animals and in surgical patients. Their work seems to indicate that obstruction of the appendical lumen is the prime factor in the pathogenesis of the disease. They have shown that the appendix has a secretory function and that with obstruction it becomes analogous to a "closed loop." Continued secretion with obstruction may raise the intraluminal tension to the level of the systolic blood pressure. The anoxic appendical wall then provides an ideal medium for invasion by colonic bacteria in the secondary, or infective, phase of the disease.

Obstruction of the appendical lumen may be produced by several pathologic entities. Wangensteen,[2] was able to demonstrate the presence of Gerlach's mucosal fold in 80 per cent of over 500 cadavers and also, by microscopic study of the cecoappendical junction, the existence of a sphincter-like group of circular muscle fibers at the appendical base.

These two factors may possibly offer some resistance to the outflow of luminal secretions, but fecaliths appear to be the most positive cause of appendical obstruction. These concretions were found in 40 per cent of all appendixes in The Children's Hospital series on pathologic examination. In 66 per cent of specimens exhibiting perforation, fecaliths were present. The actual figure of incidence is probably much higher than this. Obviously, by the time of operation the fecalith may already have been extruded into the cecum or into the general peritoneal cavity through a perforation. Unfortunately, the presence or absence of a fecalith in the peritoneal cavity has not been specifically recorded in most of the operative notes in our group of cases.

Another factor which may predispose to the development of acute appendicitis in children is infection of the appendix with pinworms (Oxyuris vermicularis). These parasites were present in the appendixes of 60 patients presenting the clinical picture of acute appendicitis in this five and a half year period. In 25 of these patients there was acute inflammation of the appendix, with perforations in 4 instances. The appendixes of the remaining 35 children, who had a typical clinical picture of appendicitis, showed no gross or microscopic inflammatory changes, and their cases are not included in this series. The relationship of pinworms to appendicitis was discussed by Botsford, Hudson and Chamberlain [3] in 1939, but no definite conclusions as to etiologic connection could be formed. Nevertheless, it is possible that a bolus of pinworms may occasionally obstruct the appendical lumen in the same way as a fecalith does.

PATHOLOGIC SEQUENCES OF APPENDICITIS IN CHILDHOOD

With the establishment of obstruction of the appendical lumen the familiar changes of appendicitis take place in a well defined pattern. Increased intraluminal tension produces anoxia with edema of the mucosal layer and swelling of lymphoid follicles, frequently resulting in necrosis and ulceration of mucosa, especially at the points where follicles approach the surface. Bacteria present in the mucosa and luminal secretions may then penetrate the mucosal barrier, spreading readily in the loose submucosa and inciting a diffuse inflammatory process. The infection then passes through the muscularis along penetrating vessels and into lymphatics, as described by Aschoff,[4] with further diffuse spread in the subserosal layer. Increased venous congestion results in transudation of fluid into the peritoneal cavity. This is enhanced by spread of inflammation to the serosal layer of the appendix and adjacent peritoneal surface, with the establishment of a periappendicitis. Perforation occurs with suppurative necrosis of the wall of the appendix or as a result of gangrene due to vascular thrombosis.

In general, it seems that this progression of changes leading to perforation is much more

2. Wangensteen, O. W.; Buirge, R. E.; Dennis, C., and Ritchie, W. P.: Ann. Surg. **106**:910, 1937. Wangensteen, O. W., and Dennis, C.: ibid. **110**:629, 1939.

3. Botsford, T. W.; Hudson, H. W., Jr., and Chamberlain, J. W.: New England J. Med. **221**:933, 1939.

4. Aschoff, L.: Appendicitis, London, Constable & Co., Ltd., 1932.

rapid in children than in adults. Of greater importance is the inadequacy of the "walling-off" mechanism in children with perforative appendicitis. In a child the appendix is usually longer in relation to the size of the peritoneal cavity and the cecum is frequently more mobile than in an adult. These two factors permit more widespread peritoneal contamination at the time of perforation. In addition, the omentum is frequently so short, so devoid of fat and so underdeveloped in early life that it offers little or no protection against spreading peritonitis of appendical origin. In older children, as in adults, the long, fatty omental apron usually serves to localize appendical peritonitis by adherence, thus forming a protective wall which inhibits diffuse peritoneal infection and facilitates the development of a local abscess.

TABLE 3.—*Location of Pain in Relation to Type of Appendicitis*

Location	Acute Unruptured		Acute Ruptured	
	No. of Cases	Per Cent	No. of Cases	Per Cent
Periumbilical region.....	77	28.0	98	42.0
Right lower quadrant....	176	65.0	86	36.0
Generalized.....	8	3.0	44	18.0
Left lower quadrant.....	4	1.5	2	0.9
Lower portion of abdomen.....	7	2.5	3	1.3
Right upper quadrant....	..	....	1	0.4

DIAGNOSIS

Acute appendicitis is usually an easily recognizable clinical entity in later childhood, as the classic textbook features are commonly present. However, in infancy and early childhood this is unfortunately, not the case, and accurate early diagnosis may be extraordinarily difficult. Obviously, in dealing with such patients history can be obtained only from second-hand sources and is frequently of little help. The inability of an infant or young child to express clearly the presence of the pain of early appendicitis, together with misinterpretation of such manifestations of pain by parents, is likely to lead to serious delay in diagnosis. Fretfulness, irritability, refusal of feedings, disturbed sleep and screaming spells may be present for several hours before the parent becomes aware that the child objects to handling of the abdomen.

When pain can be accurately localized, it is most apt to begin around the umbilicus, with a later shift to the right lower quadrant of the abdomen on the development of periappendicitis. This sequence may be difficult to elicit, as children are frequently vague in describing their symptoms. With the development of peritonitis, pain becomes generalized and severe. Table 3 compares the presenting location of pain at the time of hospitalization in simple and in perforative appendicitis.

Nausea and vomiting are constant symptoms in childhood appendicitis and usually occur subsequent to onset of pain. Only 13 children in this study failed to give a history of nausea. Vomiting occurred in 419 patients, an incidence of 83 per cent.

Temperatures of 100 to 101 F. usually accompany appendicitis in children and rarely exceed 102.6 F. except in patients with perforation and peritoneal involvement. Table 4 shows the relation between temperature and type of appendicitis in our patients.

The bowels are usually normal, but constipation prior to and during an attack of appendicitis is common, occurring in 26 per cent of these children. Diarrhea occurred in 83 children (16 per cent), almost all of whom had either spreading peritonitis or a pelvic appendicitis, most often with abscess, resulting in irritation of the sigmoid.

Urinary symptoms are uncommon, occurring in only 33 children in this group. Dysuria and frequency suggest that the inflamed appendix lies against the urinary bladder or over the right ureter. One child had grossly bloody urine associated with signs of appendicitis and at operation was found to have a perforated appendix localized by adherence to the dome of the bladder, with resulting secondary hemorrhagic cystitis.

TABLE 4.—*Relation Between Type of Appendicitis and Temperature*

Type	Temperature Range					
	Normal	100.6 F.	101.6 F.	102.6 F.	103.6 F.	104.6 F.
Acute unruptured...	52	146	63	20	8	4
Acute ruptured.....	10	36	61	63	46	16

Abdominal tenderness is the most consistent objective sign and unquestionably the most reliable one in the diagnosis of appendicitis, being absent in only 2 of these 506 patients. Maximal tenderness in the right lower quadrant of the abdomen was elicited in 385 children (76 per cent). In table 5 are listed the sites of tenderness in cases of simple and of perforated appendicitis.

Rectal tenderness was the second most common physical sign elicited in this series, being present in all but 15 cases of perforated appendicitis and absent in only 54 cases of simple acute appendicitis—a combined incidence of 87 per cent (table 6).

Involuntary spasm of the rectus muscle was found in 64 per cent of cases of unperforated and in 94 per cent of cases of perforated appendicitis (table 7).

We feel it important to stress that care and gentleness must be exercised to obtain satisfactory information from the abdominal and rectal examination of sick and uncooperative children. Inspection alone will reveal a great deal to the observer during the all-important period spent in gaining the child's confidence. The usual order of physical examination from head to toes must be completely altered. The examiner should first turn to the abdomen, with emphasis on gentle palpation and unhurried movement from nontender points to suspected areas of tenderness. Differentiation between voluntary and involuntary spasm in young children may be rather difficult. Considerable aid in this regard may be obtained by splinting the abdomen with the examining hand and allowing the latter to rest in one position for several minutes. After this maneuver, if resistance of the rectus muscle persists, particularly during the inspiratory phase of respiration, peritoneal irritation may be diagnosed with accuracy, even for the most uncooperative child.

After completion of the general physical examination, digital examination of the rectum should always be performed. The well lubricated finger should slowly dilate the sphincter, and palpation of the pelvis should be deferred until the patient's cooperation is regained. Because of the child's small pelvis this maneuver, especially when combined with simultaneous abdominal palpation, may reveal information not detected by abdominal examination alone. Indeed, a low-lying pelvic appendix and some appendical abscesses may be discovered only by this means (table 8).

A positive psoas sign was recorded in 63 cases in this series. Irritation of the psoas muscle may be readily apparent by a limping gait and the tendency of the child to lie with the right thigh partially flexed. Accessory methods of examination commonly used to elicit referred tenderness, such as cough and rebound tests, are of little value for young patients. Cooperation is difficult to obtain, and tenderness is almost inevitably referred to the area touched by the examiner.

A white blood cell count on admission of over 10,000 per cubic millimeter was encountered in 92 per cent of these 506 cases (table 9).

An elevation of polymorphonuclear leukocytes in the differential count was present even more consistently than was total leukocytosis (table 10).

TABLE 5.—*Site of Maximal Tenderness in 506 Cases of Appendicitis*

Location	Acute Unruptured, Number of Cases	Acute Ruptured, Number of Cases
Right lower quadrant	259	126
General	4	91
Lower portion of abdomen	8	12
Left lower quadrant	2	1
Right upper quadrant	...	2

TABLE 6.—*Location of Maximal Rectal Tenderness as Related to Type of Appendicitis*

Rectal Tenderness	Acute Unruptured, Number of Cases	Acute Ruptured, Number of Cases
Right	211	143
Right and left	6	73
Left	1	3
Not elicited	54	15

TABLE 7.—*Spasm of Rectus Muscle in Simple and in Perforated Appendicitis*

Spasm	Acute Unruptured, Number of Cases	Acute Ruptured, Number of Cases
Right rectus	164	127
Generalized	7	89
Left rectus	3	3
Absent	99	14
Right rectus, upper half	...	1

TABLE 8.—*Palpable Mass in 506 Cases of Appendicitis*

	Mass Palpated in 55 Cases by		
	Abdominal Examination Only, Number of Cases	Rectal Examination Only, Number of Cases	Combined Examinations, Number of Cases
Right lower quadrant	22	10	21
Left lower quadrant	..	..	1
Right upper quadrant	1	..	..

TABLE 9.—*Range of Leukocyte Count*

Leukocytes per Cu. Mm.	Acute Unruptured, Number of Cases	Acute Ruptured, Number of Cases
Under 10,000	25	17
10,000-15,000	86	59
15,000-20,000	80	61
20,000-30,000	50	82
Over 30,000	2	19

It should be noted that in 17 children who had leukocyte counts below 10,000 perforation with peritonitis or abscess was present while 52 children without peritoneal involvement had a leukocyte count of over 20,000.

Some degree of ketosis, as manifested by acetonuria, was almost invariably present in these patients. This can be related directly to the high incidence of nausea and vomiting in children with appendicitis and the rapid development of dehydration.

In the differential diagnosis of acute appendicitis in children one must adequately exclude gastroenteritis, constipation, nontuberculous mesenteric adenitis, acute pyelonephritis, bronchopneumonia and primary peritonitis. Rarely, acute rheumatic fever, acute poliomyelitis, diabetic coma and synovitis of the right hip may be confusing. In babies and young children intussusception and the symptoms of acute otitis media may occasionally be mistaken for appendicitis.

TABLE 10.—*Differential Count in Simple and in Perforated Appendicitis*

Per Cent of Polymorphonuclears	Acute Unruptured, Number of Cases	Acute Ruptured, Number of Cases
Under 70	13	5
70 - 80	46	35
80 - 90	76	78
90 -100	34	34

In general, abdominal pain, vomiting and slight fever should always be considered as due to acute appendicitis until proved otherwise. It is the policy of this clinic to hospitalize patients with these signs; repeated examinations are thereby facilitated, and an accurate early diagnosis can be made.

TREATMENT

There is little doubt that the treatment of acute unruptured appendicitis in children by immediate appendectomy is the procedure of choice. The management of early appendicitis is well standardized and offers no problem. The mortality rate of appendectomy for early appendicitis in most clinics approaches zero. However, for perforative appendicitis with peritonitis or abscess, mortality rates remain distressingly high, particularly in children. The inadequacy of localizing factors in children with perforative appendicitis, resulting in diffuse peritoneal contamination, makes appendectomy imperative as soon as the patient's condition permits in order to remove the source of infection. Delayed operation, or Ochsnerization, results in death from peritonitis before a localized abscess can be formed in a prohibitively high percentage of cases in childhood. Conversely, if early operation is employed for children with appendical peritonitis without adequate preoperative preparation, many unnecessary deaths occur from toxemia.

It is the policy of this clinic [5] to individualize the preparation of children with appendical peritonitis or abscess for operation, delaying appendectomy only for the brief period necessary to obtain the maximum benefit of supportive measures. For children with localized peritonitis or abscess this may be only a matter of three to six hours, but for the severely ill, extremely toxic children with neglected general peritonitis, frequently twelve to eighteen and occasionally twenty-four hours of supportive therapy are indicated before appendectomy is justified.

Since dehydration and ketosis are so common, even in children with early appendicitis, preoperative intravenous administration of dextrose in isotonic solution of sodium chloride is almost routine in this clinic. For the seriously ill children with peritonitis a constant intravenous drip is instituted for the infusion of plasma, as well as dextrose and saline solutions. Sodium sulfadiazine is given parenterally in an initial dose of ½ grain (0.03 Gm.) per pound (0.5 Kg.) of body weight. Fowler's position, constant gastric suction by the Wangensteen method, morphine in three to four hourly doses and high concentration (95 per cent) oxygen complete the therapeutic regimen.

The decision as to time for operation in these cases is based on the response of the patient to this therapy with improvement in his clinical status as evidenced by his general appearance, a significant reduction in temperature and, of greatest importance, a 20 to 30 per minute decrease in pulse rate with improvement in its quality (see chart).

Although most of our patients with peritonitis responded satisfactorily to these supportive measures in six to twelve hours, a few have shown little change in their extreme toxicity even after twenty-four hours of treatment. When one is faced with this problem, considerable clinical judgment is required, and it is essential to recall that removal of the ruptured appendix offers the child the greatest assistance in overcoming his infection. However, in rare cases the moribund state of the child may preclude any more extensive procedure than simple incision and drainage of the abdomen under local anesthesia. While this procedure is not ideal,

5. Ladd, W. E., and Gross, R. E.: Abdominal Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1940.

the temporary avenue of escape which it offers to peritoneal exudate is of far greater advantage in young patients than is prolonged Ochsnerization alone.

All the 506 patients with acute appendicitis observed in The Children's Hospital between January 1939 and July 1944 were operated on. The appendix was removed at the initial operation in 472 cases (93 per cent), while in 34 cases (7 per cent) it was deemed inadvisable to do more than incision and drainage. Of these patients 30 were later admitted for interval appendectomy.

The choice of incision for appendectomy in children depends on the size of the child and the type of appendicitis. Because of the small area which presents between the linea semilunaris and the anterior superior iliac spine in young children, the McBurney incision may give inadequate exposure, particularly of pelvic appendixes. In this clinic use of this incision is largely restricted to older children with early appendicitis and well localized tenderness of the right lower abdominal quadrant or retrocecal appendicitis and as a means of extraperitoneal drainage of an appendical abscess.

The right rectus incision gives excellent exposure of the appendix in children with minimum retraction. Retraction of the rectus muscle is preferable to splitting of the belly of the muscle, since it insures a stronger wound. As medial retraction easily damages the nerve supply of the muscle, we have recently employed lateral rectus retraction almost exclusively. If drainage is indicated, the tubing may be led out through a stab wound in the lower portion of the belly of the rectus muscle. Types of incision used in this series are recorded in table 11.

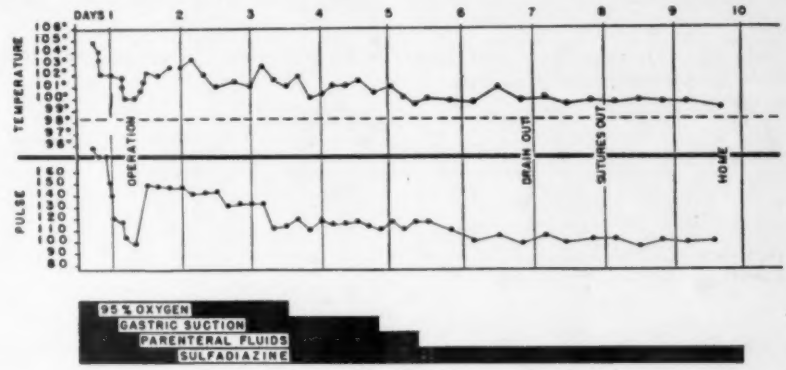
It is the policy in this clinic to invert the appendical stump into the cecum with a purse string suture of silk, except in the presence of an edematous, markedly inflamed and friable cecal wall.

We feel that drainage of the pelvis has a distinct usefulness for children with peritonitis of appendical origin. There has been considerable argument in surgical literature between the proponents of peritoneal drainage and the opponents of this practice, beginning with the work of Yates [6] in 1905. In recent years there has been a distinct trend toward the more infrequent use of drains, particularly since the advent of chemotherapy. While it is admittedly impossible to drain the entire peritoneal cavity, except temporarily, it is the opinion of this clinic that a drain left in the pelvis in cases of appendical peritonitis has two distinct advantages: 1. It allows a temporary means of escape of exudate whose absorption would mean more fever and toxicity in the immediate postoperative period. 2. A sinus is formed into which local collections of pus in the pelvis or iliac fossa may subsequently rupture or be evacuated, and thus additional operative procedures and delayed convalescence can be avoided. In table 12 the operative procedures employed in the 506 cases are listed.

The use of intraperitoneal drains has been opposed by many authors on the ground that drainage increases peritoneal adhesions and con-

TABLE 11.—*Types of Incision Used in 506 Cases of Appendicitis*

Incision	Number of Cases
Right rectus, lateral retracting	206
McBurney	120
Right rectus, medial retracting	100
Rectus splitting	78
Rockey	1
McBurney, left	1
Left rectus, medial retracting	1



Clinical course of a case of severe appendical peritonitis. The patient was a 24 month old child weighing 24 pounds (10.9 Kg.).

6. Yates, J. L.: Surg., Gynec. & Obst. **1**:473, 1905.

sequently results in a greater incidence of intestinal obstruction following peritonitis. While we do not deny this possibility, our experience has not confirmed it, and we feel that other factors are more significant in this regard. Among these must be listed operative trauma to visceral and parietal peritoneum (rough handling, glove powder, heavy retraction, dry gauze and so forth) and localized intraperitoneal abscesses. The incidence of the latter is reduced by a thorough aspiration of pus at the time of operation, gently separating fibrinous adhesions by the wet, gloved finger so as to release pocketed exudate for removal by suction. In this clinic we use soft, pliable rubber drains with a central core of gauze (Penrose). The drain is always led down into the pelvis along its lateral wall and should never be placed among loops of bowel or against the appendical stump.[5] Although we have tended to use drainage less often in recent years, we feel that results obtained in the treatment of appendical peritonitis in this hospital offer substantial support to the rationale of this mode of therapy.

Table 13.—*Postoperative Abscesses in Relation to Treatment*

Operative Procedure	Cases	Pelvic Abscess	Wound Abscess	Subdiaphragmatic Abscess
Appendectomy	233	—	2	..
Appendectomy with drain	33	5	..	..
Appendectomy with local and/or parenteral sulfonamide therapy	36	4	3	..
Appendectomy with drain and parenteral sulfonamide therapy	97	17	6	..
Appendectomy with drain and local and parenteral sulfonamide therapy	73	6	5	1
Incision and drainage with parenteral and/or local sulfonamide therapy	29	2	1	2
Incision and drainage	5	..	..	..
Totals	506	34	17	3

Table 12.—*Operative Procedures Employed for Various Types of Appendicitis*

Type	Appendectomy, Number of Cases	Appendectomy with Drain and Sulfonamide Compounds Locally, Number of Cases	Appendectomy with Drain, Number of Cases	Incision and Drainage, Number of Cases	Appendectomy with Sulfonamide Compounds Locally, Number of Cases
Acute unruptured	247	6	13	..	6
Acute ruptured with peritonitis	6	61	103	11	5
Acute ruptured with abscess	1	6	18	23	..

Intraperitoneal administration of sulfonamide compounds was used in 72 appendectomies for perforative appendicitis with abscess or peritonitis. In 67 of these cases a drain was also employed. Sulfadiazine was used in 50 cases, sulfanilamide in 17 and sulfathiazole in 5. Five patients with ruptured appendixes were treated by appendectomy with intraperitoneal applications of a sulfonamide compound and no drainage, and 6 by appendectomy with no local drug or drain. All these 11 patients received additional chemotherapy orally and/or parenterally in the postoperative period.

In table 13 we have tabulated the postoperative wound infections and postoperative peritoneal abscesses which have been encountered in this series. While no definite statistical conclusions can be drawn from these figures, it seems that in our cases the intraperitoneal use of sulfonamide compounds offered no great advantage over the oral or parenteral use of sulfathiazole or sulfadiazine in reducing the incidence of these complications.

When a well localized appendical abscess presents as an abdominal mass at the time of hospitalization, direct drainage should be employed whenever possible. Incision should be centered over the presenting mass and the abscess entered directly without an opening into the uncontaminated peritoneal cavity. This may usually be accomplished through a McBurney incision, but in a few instances the position of the abscess may be such that a rectus incision offers a more direct approach. The appendix is removed if easily available, and the cavity is drained. However, when the appendix is incorporated in the wall of the abscess and cannot be removed without destruction of the inflammatory barrier, with subsequent generalized peritoneal infection, simple drainage should be instituted and appendectomy deferred for about six months.

The postoperative period following appendectomy for acute nonperforative appendicitis in children presents few problems. We have used chemotherapy postoperatively only when smears or cultures of the peritoneal fluid taken at operation have revealed organisms. By the routine use of Wangensteen's gastric suction for six to twelve hours after operation vomiting and the

associated danger of aspiration in young children may be successfully obviated.

The importance of a well planned postoperative regimen for children with appendical peritonitis cannot be overemphasized. Rest for the patient and, insofar as possible, for the gastrointestinal tract is essential for resolution of peritoneal inflammation. Morphine is given in adequate doses every three to four hours. Constant gastric siphonage by the Wangensteen method with an inlying Levine tube in older children or a urethral catheter in babies should be used routinely to prevent nausea, vomiting and the distention of adynamic ileus. Miller-Abbott tubes are usually not necessary and because of their size have a limited usefulness for children. Fluids for parenteral administration are best given by a constant intravenous drip through a 19 or 20 gage needle, which will usually be tolerated for eighteen to twenty-four hours without danger of thrombophlebitis. Occasionally it may be necessary to cannulate a vein directly. It is our feeling that bone marrow infusions, with their inherent danger of osteomyelitis, periosteal stripping and subsequent sequestration, should rarely be used. Marrow infusions are advocated chiefly for children under 2 to 3 years of age, but we feel that it is safer and simpler to give repeated infusions by scalp veins in this age group, short-beveled 24 gage needles being used and the fluid administered under slight pressure. Solutions of 5 or 10 per cent dextrose in water or isotonic solution of sodium chloride are used most commonly in combating the ketosis, dehydration and fluid losses in these children.

Precautions must be observed to avoid overloading the child with excessive saline solutions. An amount of isotonic solution of sodium chloride may be given roughly corresponding to Wangensteen drainage; but if prolonged suction is required, determinations of blood chloride and serum protein levels are necessary to indicate more accurately the type of fluid required. Infusions of plasma are indicated for hypoproteinemia and transfusions of whole blood if there is anemia as well. It is felt that repeated small transfusions of fresh whole blood are helpful for critically ill children with peritonitis. If suction and parenteral administration of fluids are necessary for more than two or three days, solutions of amino acids should be added to the parenteral feeding regimen. By this means, protein deficiency from inadequate intake and loss in peritoneal exudate may be compensated.

It is important to maintain a strict chart of intake and output in order to regulate fluid balance. Daily requirements for parenterally administered fluids average 2.5 to 2 ounces (75 to 60 cc.) per pound (0.5 Kg.) for babies under 2 years and 1.5 ounces to 1 ounce (45 to 30 cc.) per pound for children over 2 years of age.

Chemotherapy was administered postoperatively in 87 per cent of the cases of perforated appendicitis in this series. Sulfathiazole was used in 82 cases and sulfadiazine in 99. Both drugs seemed effective in diminishing postoperative toxicity, shortening convalescence and reducing complications, but fewer reactions to the drug were encountered with sulfadiazine. Sodium sulfadiazine should be administered subcutaneously or intravenously in doses of ½ to ¾ grain (0.06 to 0.09 Gm.) per pound per day until gastric suction is discontinued, when the drug is given by mouth. It is our practice to continue chemotherapy for these children after operation until all signs of infection have abated, as evidenced by a normal clinical chart and white blood cell count, a cessation of discharge from the drain sinus and negative results of rectal examination.

We have routinely employed Fowler's position for all patients with peritonitis in this series and feel that it is helpful in evacuating exudate when drainage has been used. Despite certain theoretic objections to the usefulness of this position in enhancing the pelvic localization of exudate, it is interesting to note that in only 3 cases did subdiaphragmatic abscess occur in our patients. Ninety-five per cent oxygen has been found extremely useful for children with diffuse peritonitis as a means of preventing or of reducing the distention of paralytic ileus. Hot abdominal poultices and rectal tubes have been used routinely at two to three hourly intervals in the postoperative period for a similar purpose. Heat to the abdomen probably has its greatest benefit in increasing the patient's comfort and decreasing the incidence of infection in the wounds.

In table 14 we have compiled statistics on postoperative treatment in our cases of appendical peritonitis or abscess. These show a trend toward a more standardized regimen in recent years.

COMPLICATIONS

The various complications encountered in these 506 cases are listed in table 15. Pelvic abscess was the most common postoperative complication, occurring in 12 per cent of the 234 cases of perforating appendicitis in this series. In no instance was operative drainage of a pelvic abscess required. Treatment consisted in the resumption of chemotherapy if this had been discontinued and the use of hot rectal irrigations.

With this regimen 2 abscesses drained spontaneously by rectum, while the remainder were either resorbed or evacuated via the drain sinus. There was no mortality in this group. We have been unable to find in the literature a comparable group of cases of appendical peritonitis treated without drainage in which figures on this complication were listed, but we feel that our experience in this regard illustrates the value of drainage.

Intestinal obstruction was encountered subsequent to operation in 7.3 per cent of the cases of perforated appendicitis. In 17 cases this complication appeared during the first or second week after operation, while in 2 cases late obstruction occurred five and twelve months respectively following peritonitis. Early intestinal obstruction is usually a result of friable, fibrinous adhesions due to a plastic exudate between loops of bowel. In the earlier cases of this series Witzel enterostomy was usually done without delay, but this has been rarely necessary in recent years. Resorption of exudate with relief of the obstruction will take place in most instances within a few days if decompression of the distended bowel is accomplished by constant gastric siphonage or the use of a Miller-Abbott tube. High concentration oxygen is also of definite value in this regard. For late intestinal obstruction laparotomy and lysis of adhesions is clearly indicated. In table 16 we have recorded the time of onset and type of treatment used in the cases in this series in which intestinal obstruction developed. No deaths occurred as a result of this complication.

The secondary operations which were performed in this entire group of 506 cases are listed in table 17. An effort was made to readmit every child who had simple drainage of an

TABLE 14.—*Postoperative Treatment of Perforated Appendicitis*

Year	Total No. of Cases	Fluids Parenterally		Sulfonamide Compounds		Suction		Oxygen (95 per Cent)	
		No. of Cases	Average Duration, Days	No. of Cases	Average Duration, Days	No. of Cases	Average Duration, Days	No. of Cases	Average Duration, Days
1939	27	24	4	8	4.7	19	2	9	3
1940	30	29	6.5	25	9	27	4	11	3.3
1941	46	46	4	45	6.4	42	3.2	20	1.7
1942	48	48	3	47	12	42	2	6	1.7
1943	58	57	3	50	8	52	2	7	2.4
1944	20	20	3.5	20	10.3	19	2.6	8	2.6

TABLE 15.—*Postoperative Complications in Relation to Type of Appendicitis*

Complication	Acute Unruptured, Number of Cases	Acute Ruptured, Number of Cases	Operative Treatment, Number of Cases
Pelvic abscess	4	39	None
Intestinal obstruction	3	17	12
Pneumonia	3	15	None
Wound abscess	5	12	6
Fecal fistula	1	3	None
Contagious disease	3	1	None
Evisceration	None	3	3
Atelectasis	3	None	None
Bacteremia	1	2	None
Iliac abscess	None	2	2
Pyelonephritis	1	1	None
Wound hernia	None	2	2
Subphrenic abscess	None	3	3
Ether convulsions	None	2	None
Retroperitoneal abscess	1	1	1
Thrombophlebitis	1	None	None
Myocardial failure	None	1	None

TABLE 16.—*Time of Onset and Treatment of Intestinal Obstruction*

Patient	Year	Type of Appendicitis	Onset of Obstruction (Postoperative)	Treatment	Result
1	1939	Perforated, with general peritonitis	5 days	Witzel enterostomy	Well
2	1939	Perforated, with general peritonitis	5 days	Witzel enterostomy	Well
3	1939	Perforated, with abscess	3 days	Witzel enterostomy	Well
4	1939	Acute, unruptured	9 days	Lysis of adhesions and Witzel enterostomy	Well
5	1939	Perforated, with general peritonitis	5 days	Witzel enterostomy	Well
6	1939	Perforated, with general peritonitis	5 days	Witzel enterostomy	Well
7	1940	Perforated, with abscess	3 days	Miller-Abbott suction (8 days)	Well
8	1940	Acute, unruptured	11 days	Miller-Abbott suction 5 days, then lysis of adhesions	Well
9	1940	Perforated, with general peritonitis	12 days	Gastric suction (3 days)	Well
			(*a*) 9 days	(*a*) Witzel enterostomy	Well
10	1941	Acute, unruptured	(*b*) 5 mo.	(*b*) Lysis of adhesions	Well
11	1941	Acute, unruptured	8 days	Suture of evisceration; lysis of adhesions	Well
12	1941	Perforated, with local peritonitis	8 days	Gastric suction (3 days)	Well
13	1942	Perforated, with general peritonitis	11 days	Gastric suction (3 days)	Well
14	1943	Perforated, with abscess	17 days	Gastric suction (3 days)	Well
15	1943	Perforated, with local peritonitis	12 mo.	Lysis of adhesions	Well
16	1944	Perforated, with abscess	On admission	Incision and drainage of abscess; Witzel enterostomy	Well
17	1944	Perforated, with general peritonitis	16 days	Gastric suction (1 day)	Well
18	1944	Perforated, with local peritonitis	7 days	Miller-Abbott suction (10 days)	Well

appendical abscess or of an overwhelming appendical peritonitis without appendectomy about six months after the initial illness for interval appendectomy.

MORTALITY

There were 8 deaths in this series of 506 cases, an over-all mortality rate of 1.58 per cent. Only 1 death occurred in the 272 cases of acute unruptured appendicitis. This patient was a 10 year old Negro boy who had symptoms of abdominal pain, chills and swinging fever for nine days before operation. An acutely inflamed, unruptured appendix was found in a high retrocecal position with no peritonitis but with well established pylephlebitis. The postoperative course was extremely stormy, with daily rises in temperature to 105 to 107 F., and the child finally died on the forty-fifth day in the hospital.

TABLE 17.—*Secondary Operations in 506 Cases of Appendicitis*

Operative Procedure	Number of Cases
Interval appendectomy	30
Witzel enterostomy	8
Lysis of adhesions	4
Drainage of wound abscess	6
Drainage of subphrenic abscess	3
Drainage of retroperitoneal abscess	1
Drainage of iliac abscess	2
Suture of evisceration	3
Incisional herniorrhaphy	2

TABLE 18.—*Relation of Age of Patient and Type of Appendicitis to Mortality*

Total cases in series, 506; over-all mortality rate, 1.58 per cent.

Age Group, Years	Acute Unruptured Appendicitis: Number of Cases	Deaths	Mortality, per Cent	Acute Ruptured Appendicitis: Number of Cases	Deaths	Mortality, per Cent
0 - 1	1	0	0.0	0	0	0.0
1 - 2	0	0	0.0	22	3	13.6
2 - 4	15	0	0.0	59	3	5.1
4 - 6	37	0	0.0	42	0	0.0
6 -12	195	1	0.5	109	1	0.9
12 -16	24	0	0.0	2	0	0.0
Total	272	1	0.36	234	7	2.99

The 7 deaths in our 234 cases of perforated appendix all occurred in children with generalized peritonitis—a mortality rate of 2.99 per cent. Four of these children had pneumonia in addition to peritonitis. Two other patients died with "ether convulsions" and hyperpyrexia immediately after operation. In retrospect, in neither of these cases had there been adequate preoperative preparation, and both children were operated on while they were under ether anesthesia and had temperatures over 104 F. and pulse rates over 150. With adequate preoperative preparation, these deaths might easily have been prevented. The last fatality occurred in a child in whom a bacteremia due to Bacillus coli developed after appendical peritonitis with a terminal B. coli endocarditis.

TABLE 19.—*Relation Between Duration of Symptoms and Type of Appendicitis*

Duration of Symptoms	Acute Unruptured Appendicitis	Acute Ruptured Appendicitis	Incidence of Perforation, per Cent
0 to 12 hours	67	2	3
12 to 24 hours	116	36	23
24 to 36 hours	28	28	50
36 to 48 hours	24	43	64
2 to 3 days	11	19	63
3 to 4 days	14	34	70
4 to 5 days	5	11	70
5 days and over	7	61	90

TABLE 20.—*Duration of Symptoms in Relation to Mortality*

Duration of Symptoms Before Operation	Cases	Deaths	Mortality, Percentage
0 to 12 hours	69	0	0.00
12 to 24 hours	152	1	0.65
24 to 36 hours	56	0	0.00
36 to 48 hours	67	1	1.49
2 to 3 days	30	2	6.66
3 to 4 days	48	1	2.08
4 to 5 days	16	1	6.25
5 days and over	68	2	2.94

TABLE 21.—*Relation Between Type of Appendicitis and Mortality Rate at the Children's Hospital, Boston, from 1928 to 1939 and from 1939 to 1944*

Type of Appendicitis	Cases	Deaths	Mortality, Percentage
1928 to 1939			
Acute, unruptured	475	2	0.42
Acute, ruptured with local or diffuse peritonitis	373	24	6.43
Totals	848	26	3.06
1939 to July 1944			
Acute, unruptured	272	1	0.36
Acute, ruptured with local or diffuse peritonitis	234	7	2.99
Totals	506	8	1.58

In table 18 we have presented the relation between age of patient, type of appendicitis and mortality rate in our patients. For children under 4 years of age, the mortality rate was 6.2 per cent, as compared with 0.48 per cent for children over 4 years. This discrepancy must be attributed to the more rapid development of generalized peritonitis and to the greater difficulty encountered in making early diagnosis for the younger children.

The relation between duration of symptoms and type of appendicitis is recorded in table 19. In table 20 the duration of symptoms is compared with the mortality rate. It is discouraging to realize that almost one fourth of our patients were hospitalized only after three days or more of symptoms. This represents the same proportion of cases of neglected appendicitis that was encountered in the children with appendicitis treated in this clinic between 1928 and 1939 and would seem to indicate that there has been no advancement in the early recognition of the disease in children by practitioners or by the lay public.

A comparison of mortality rates in the present series and those of the preceding ten year period is presented in table 21. It is interesting to note that the over-all mortality rate has been reduced by approximately 50 per cent and that the recent percentage of fatalities in cases of perforated appendicitis is actually lower than the over-all rate of the earlier group of patients.

It seems apparent that that the accomplishment of improvement in the treatment of appendicitis, particularly of the perforative type, must be attributed to multiple factors and not merely to the use of sulfonamide drugs. These factors include more adequate preoperative preparation; early appendectomy, as soon as general toxicity is reduced; a better standardized postoperative regimen, including routine gastric suction, Fowler's position and parenteral administration of fluids, with the addition of sulfadiazine in cases of perforation. As a result of these factors, no deaths occurred in this clinic in 236 cases of appendicitis during the last three years of this observation.

SURGICAL TREATMENT OF LYMPHEDEMA

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CINCINNATI

The question of the treatment of elephantiasis has been a puzzling one for many years. The decision as to method is not greatly influenced by consideration of the etiologic factors of the disease, which is always due to an obstruction of the normal lymph channels and subsequent swelling of the extremity. For the purpose of classification, lymphedema may be stated to be due (1) to filariasis; (2) to inflammatory or scar tissue obstructing the normal lymph channels, as occurs commonly after radical operations on the breast, and (3) to some obscure lesion that obstructs the lymph channels. The last, which may be called idiopathic, is a very small class. The kind following phlegmasia alba dolens is not considered here, as this is far more venous than lymphatic in origin.

The problem of surgical treatment in cases of lymphedema has long taxed the ingenuity of surgeons. The subject assumes greater importance with the possibility of a large number of cases occurring in our troops in the tropical theaters of war. Several operative procedures have been suggested and tried. The Kondoleon operation has had the greatest vogue and has given more or less indifferent results. The Kondoleon operation, if done properly, is an operation of considerable magnitude. With the meager results so far obtained, it is doubtful whether this operation has great applicability.

An operation suggested by Handley [1] in 1908 should be given further trial. The principle is to bury long silk sutures in the edematous area to serve as permanent lymph channels and to conduct the lymph away from the infected area to normal lymph channels.

Recently I have used a modified Handley operation in 2 cases, and the results were gratifying. The result in the first case is startling.

REPORT OF CASES

CASE 1.—A nurse, aged 24, six months before she consulted me, while at work, suddenly noticed a swelling of the left hand, without injury and with no sensation of pain. This swelling increased in size, until it looked as though she were wearing a boxing glove on the left hand. She had been to various clinics in an attempt to remedy this condition. Her treatment had included massage and rest in bed, with elevation of the hand. An attempt was made to block the brachial plexus, with the idea that it was of neurogenic origin. Another attempt was made to inject the brachial artery, with the idea that there was some arterial disease. The swelling of the hand was so great that there was practically no motion of the fingers, and the roentgenogram showed atrophy of all the bones of the hand. The patient had lost 50 pounds (22.7 Kg.) and was in a state of extreme depression.

On Sept. 26, 1944 a modified Handley operation was done. Double strands of no. 1 nylon were used in place of silk, as it was thought that this would be less irritating and remain in the tissues indefinitely. A small transverse incision, which can be seen in the photograph, was made just distal to the first phalanx of the ring finger, and through a specially constructed long probe, the suture was passed through several small incisions, under the superficial fascia, to the normal areolar tissue in the cubital fossa. The small incisions were closed with fine silk. On the day following the operation, it was apparent that the swelling, not only of the ring finger but of all the fingers, had begun to recede, and six weeks later the hand was apparently normal. At no time was there secretion from the wounds. After the

Fig. 1 (case 1).—Normal and abnormal hand before operation. *A*, lateral view; *B*, dorsal view.

1. Handley, W. S.: Lymphangioplasty, Lancet **1**: 783, 1908.

disappearance of the swelling, the function of the hand was improved by physical therapy. Within two months the hand was completely normal.

At present, seven months after operation, the patient is back at work as a nurse and has made application to join the Navy; she has been assured by the medical examiner that she will be accepted.

Fig. 2 (case 1).—Dorsal view of both hands about four weeks after operation.

Case 2.—A 63 year old woman was operated on eight years ago for carcinoma of the left breast, The operation was followed by edema of the left arm, which increased in severity. At the time of examination the arm was almost useless, because of the indurated, brawny skin, which had marked pigskin dimpling. She could not raise her arm above the angle of 33 degrees, nor could she completely flex her elbow.

On March 22, 1945, an operation was performed according to the same technic. In this case, two sets of sutures were used, one anterior and one posterior, the posterior extending from the wrist to the subscapular fossa and the anterior extending from the wrist to the normal areolar tissue over the sternum. The improvement was almost immediate. At the time of writing, one month after operation, the woman has complete use of her arm and the skin has become soft and pliable. At the time of operation, the involved arm was 1½ to 2 inches (3.8 to 5 cm.) greater in girth measurement both in the forearm and in the upper arm than the other arm; at present the measurements of the two arms are the same.

Fig. 3 (case 1).—Dorsal view of both hands six months after operation.

INTERNAL DERANGEMENTS OF THE KNEE JOINT

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This paper reports a consecutive series of 190 arthrotomies of the knee joint done on military personnel in the Station Hospital, Camp Roberts, Calif., within a two year period. The patients operated on were in one sense selected, because operation was not advised or done if it appeared that the patient was unstable and had not the will to get well. At least an equal number of patients with affected knee joints were not operated on and have since been reclassified or discharged from service. Many of these patients had deranged joints before their entry into service and had been advised by their own physicians not to have operations. It is amazing how widely the misunderstanding has been implanted that "if the joint is opened and the water runs out the knee will be dry and stiff."

In this series the follow-up is incomplete but for practical purposes it is sufficient to permit estimation of definite results. None of the patients were seen more recently than six months before the time of writing. When the training period of three or four months is completed in Camp Roberts, the soldiers are usually moved to another camp, although many are retained for much longer periods. When a man has lost time during the training cycle, he is put into another unit and goes through the complete course, in some cases repeating part of the previous training. Almost without exception the follow-up period is at least three months, with the soldier doing the same regular duty as the other, normal men. The patients for whom reclassification was necessary will be considered later under analysis of specific types of injuries. Many patients have been followed for one year or more in this camp and through reports from other hospitals and letters. Some men have written from overseas.

DIAGNOSIS

The subject of diagnosis is so well covered in the various textbooks and journals that further comment is superfluous, except a caution against the asking of leading questions when the examiner is taking a history. The patient should be allowed to tell his own story and should always be given a choice in answering; that is, the examiner should ask, "Do you have trouble in bending or straightening the knee, or what is the difficulty?" Many patients say that the knee is "locked," but it is found with questioning that motion was not blocked at all until severe swelling occurred. A point to emphasize is that in case of acute injury the roentgenogram appears normal. One should never tell the patient that the knee has no internal articular injury because the roentgenogram is normal. If the history and examination are otherwise indicative of injury, operation should be done, and the surgeon should not be content with simple inspection of the opened joint. Gentle traction on the cartilage or exploration with a blunt hook usually demonstrates a posterior tear. Mistaken diagnoses will be made in any series of cases. In 6 cases of the present series there was no tear of cartilage but the patients had hypertrophic fat pads, which required excision.

TABLE 1.—*Analysis of One Hundred and Fifty-Five Cases of Torn Cartilage*

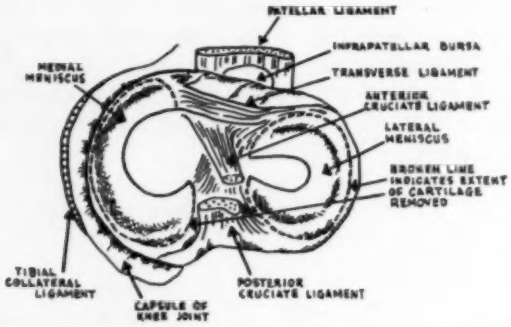
Type of Tear		Location of Tear		
			Medial	Lateral
Anterior	16	Right	79	2
Posterior	24	Left	69	5
Bucket handle	80		—	—
Lateral	29		148	7
Transverse	6	Lateral tears constitute 4.73 per cent of the total number.		
	155			

TREATMENT

In all cases the patient was taught exercises for the quadriceps muscle before the operation and was impressed with the idea that his cooperation was the deciding factor in a successful operation. In cases in which the patient exercises well after operation, the normal physiologic action is maintained and he does not suffer weakness and relaxation of the knee. One preparation of the skin is done on the day before the operation, and the whole extremity is wrapped in sterile towels. Another preparation is done while the patient is on the table. Spinal anesthesia is used in all cases. The legs hang over the end of the table with the knees at a right angle. The operator sits on a stool and the leg to be operated on can be grasped by the surgeon's own knees; this permits traction and rotation of the leg as desired.

This position is a great help in exposure of the joint and also obviates the need for one assistant. The short straight parapatellar incision is used and provides good exposure. In 3 cases, bucket handle tears of both medial and lateral meniscuses in the same knee were removed easily through one incision. If the lower pole of the incision does not sever the inferior medial articularis genu artery, there is much less postoperative swelling of the knee.

There is a difference of opinion concerning whether to remove simply the torn portion of the cartilage or the entire meniscus. In repair of bucket handle tears, the torn portion is removed, and the border of the remaining portion is cleanly excised without removal of the entire cartilage. For other types of tears, most of the meniscus is removed, but a small rim is left. One should define the term when total removal is advised and indicate just what is done. The drawing shows



Anatomic relations of structures within the knee joint.

relations of structures within the knee joint. Anatomically, each meniscus, medial and lateral, is attached by its thick peripheral border to the deep aspect of the joint capsule. In addition, the peripheral borders of the cartilages are attached to the margins of the tibial condyles by short fibrous bands, known as coronary ligaments. The peripheral margin of the medial meniscus is firmly adherent to the broad and powerful tibial collateral ligament. Thus one should excise the meniscus at least flush with the capsule, leaving a rim, instead of attempting to separate it completely from its attachments.

In 2 cases torn cartilages in both knees were removed at one operation. The patients recovered as quickly and as perfectly as the average patient who has had only one knee operated on. No conclusions can be drawn from 2 cases, but because of them it may be assumed that the procedure is not contraindicated, and I shall not hesitate to repeat it for future patients.

The joints are irrigated with sterile isotonic solution of sodium chloride and closed in layers with fine interrupted silk sutures. In no case have sulfonamide drugs been used locally or by mouth, and no infections of any sort have occurred. In my opinion chemicals are not indicated for clean surgical wounds.

The postoperative care of patients with such injuries in former years was too prolonged. Granted that a primary principle of surgery is rest, yet it has been clearly shown that the quadriceps muscle, despite its bulk, atrophies quickly. When atrophy is extreme, it is almost impossible for the muscle to regain the pretraumatic status, and the knee joint is left relaxed and unstable. Recent reports emphasize the need for exercises of the muscles before and after surgical intervention, but, in addition, it is essential that the patient begin early weight bearing, except those with initial severe hemorrhage and significant reaction. In many cases of this series there was no form of immobilization used; but for patients with noticeable swelling and sanguineous joint fluid a dressing of cotton wadding and Ace bandage was applied for two to five days. Table 2 demonstrates the time required for the return of function.

TABLE 2.—*Results of Treatment in One Hundred and Fifty-Five Cases of Torn Cartilage: All Types*

	Age, Yr.	Splint, Days	Walk, Days	90 Degree Flexion, Days	Squat on Heels, Days	Duty, Days
Averages.....	25.28	2.74	5.98	8.72	18.05	24.97
Extremes.....	18 to 42	0 to 5	2 to 11	3 to 15	7 to 35	9 to 60

The interval between injury and operation averaged 1.85 years.

In the cases of early injury splinting was routinely carried out for five days, and weight bearing was permitted after seven days. It is not logical to use an identical procedure in all cases. In the average case in which operation was performed before other damage had been done, the knee joint was not injured by removal of the cartilage. The cartilaginous surfaces of the femur and tibia were intact and articulated as before, their contours being the same without necessity of adjustment of opposing surfaces, as in an arthroplasty. There is no contraindication to early walking except that of discomfort to the patient caused by the incision. This procedure was first put into practice with the patients with bucket handle tears, and the data of table 3 are given in support of the success obtained.

With the patient walking earlier, the return of function is facilitated. The period required

for return to regular duty is generally the same, because three weeks is considered soon enough for the patient to do all the physical exercises required during training. Three of the patients were in the Medical Department, not on field duty, and were allowed to return to work at their own request as early as nine and eleven days after the operations. They had full motion of the limb and no soreness, swelling or instability. As late as eight months after operation they still had no symptoms, and they are now on foreign duty. Early walking and exercise have since been done up to the patient's tolerance, and the results show a definite improvement. As a contrast to table 2, table 4 shows the averages for the last 50 cases of all types.

TABLE 3.—*Results of Treatment in Eighty Cases of Bucket Handle Tears*

	Age, Yr.	Splint, Days	Walk, Days	90 Degree Flexion, Days	Squat on Heels, Days	Duty, Days
Average of 80 cases...	25.60	2.89	5.83	8.52	16.92	21.66
Average for last 38 cases.....	25.23	1.58	3.84	6.42	14.36	21.02

The only significant change in any factor in these 50 cases is less immobilization and earlier weight bearing in treatment; age, interval before operation and pathologic condition of the knee are essentially the same as in the whole group. These are consecutive cases, not selected any more than the entire series. The time of return to duty includes the additional days for those patients who returned to the hospital for further convalescence. It will be noted that for the entire series the extreme limit for return to duty was sixty days.

TABLE 4.—*Results of Treatment in the Last Fifty Cases*

	Age, Yr.	Splint, Days	Walk, Days	90 Degree Flexion, Days	Squat on Heels, Days	Duty, Days
Average last 50 cases.	24.84	1.08	2.76	5.58	14.24	21.00

Interval between injury and operation averaged 1.74 years.

PROGNOSIS

Judging by the average results obtained with one method of treatment, can one with reasonable certainty estimate the final result before operation? This question is most important to the surgeon. The patient is not interested in averages but wants to know positively about his own case. The surgeon is generally pessimistic about the outcome in such cases, but this pessimism is not justified in view of recent reports. An attempt is made here to determine what factors are of prognostic value. As far as this study can determine, age is no factor within the age limits of the patients that have been operated on, namely 18 to 42 years. The men over 30 were more stable and worked harder with the single purpose of rehabilitation, and all secured good stable knees and had no complaints.

The interval between injury and operation is of importance, depending on the occupation of the patient and in general on the number of recurrences of symptoms. In 155 cases these intervals were: nothing to six months, 72 cases; six to twelve months, 11 cases; one to two years, 10 cases; two to three years, 12 cases; three to four years, 12 cases; four to five years, 12 cases; five to six years, 8 cases; six to seven years, 11 cases; seven to eight years, 3 cases; nine years, 1 case; ten years, 2 cases, and 11 years, 1 case. An erosion of the cartilaginous surface of the femoral condyle directly overlying the torn portion of the meniscus is frequently seen in patients operated on several years after injury when there have been many recurrences of symptoms. This erosion is believed due to the pressure of the dislocated meniscus. The process is usually more extensive than is indicated in roentgenograms. When such changes appear, it is too late for the surgeon to accomplish a cure by meniscectomy. The mechanical impediment of blocked motion can be relieved, but a roughened articular surface and some pain remain. In a few cases erosion was seen as early as two years after injury, and it was not always shown in the roentgenogram. In the 3 cases in which the interval was from nine to eleven years, the surfaces of the joints were grossly normal and the results were good. Exact figures are not available for a large group of such patients examined in this camp but not operated on. So many patients with several recurrences of symptoms have shown moderate to extreme erosion of the femoral condyle and arthritic changes within four to five years after injury that the strong impression remains that conservative treatment for any one doing physical work

TABLE 5.—*Record of Follow-Up Observations*

Follow-Up:	6 Wk.	3 Mo.	6 Mo.	9 Mo.	12 Mo.	18 Mo.	28 Mo.	Total
By examination...	2	73	45	24	9	1	1	155
By letter.....	0	0	13	10	5	0	0	28
Final.....	1	46	58	34	14	1	1	155

is a mistake. Surgical intervention is recommended within a short time after injury, when the acute phase has receded, and without delay in cases of the bucket handle tear that is not reduced by manipulation.

In addition to these 12 patients (7.74 per cent of the total number) there were 6 who had sore knees and were given light duty for a

TABLE 6.—*Summary of Twelve Cases in Which Reclassification Was Necessary After Operation*

Case No.	Age of Patient	Interval Between Injury and Operation	Pathologic Condition of Knee	Disposition
18	22	26 days	Lateral and transverse tear; normal joint	Discharge; atrophy of disuse; psychoneurosis
30	21	6 to 7 yr.	Transverse tear; severe erosion of femoral condyle	Discharge; damage to joint
51	22	3 to 4 yr.	Lateral tear ¾ inch; normal joint	8 mo. full duty; discharge as psychoneurotic then
64	21	3 to 4 yr.	Bucket handle tear; normal joint	Reclassified; military police for 2½ yr.; pain reported; knee normal
80	27	6 to 7 yr.	Anterior and posterior tear; moderate arthritis	Reclassified; arthritis; still on duty, 15 mo.
88	22	3 to 4 yr.	Anterior and posterior tear; fat pad; normal joint	Discharge; atrophy of disuse; psychoneurosis
96	23	6 yr.	Posterior tear and hypertrophic fat pad	General duty for 1 yr.; then discharge for synovitis
126	18	2 yr.	Bucket handle tear; normal joint	Reclassified; quartermaster duty 10 mo.; no complaints
132	20	10 yr.	Bucket handle tear; was "locked" for 9 yr.	Light duty 8 mo.; discharge for unstable knee; painless
147	30	4 yr.	Posterior tear; joint normal	Reclassified; psychoneurosis
149	26	3 days	Bucket handle tear; normal joint	Full duty 3 mo.; no complaints; then acute gonococcic arthritis and discharge later
152	30	14 days	Posterior tear; normal joint	Discharge; atrophy of disuse; psychoneurosis, severe

few days at intervals over a two month period. When last examined, six months after the operation, they had no complaints or physical abnormalities. Five more had some soreness for three months but remained on full duty without loss of time; at six months they had no complaints and had normal knees. The remaining 132 (85.16 per cent) have had no complaints or physical abnormalities since they left the hospital and have been returned to duty as combat soldiers. But 7 of these (4.51 per cent) had some changes in the joints at the time of operation, and it is expected that a longer follow-up observation will show some eventual disability, although they appeared normal at six months. With the exceptions noted, the patients in this series have gone back to their full duty directly from the hospital, not being placed on light duty for three to six months. It is a mistake to allow patients to remain long inactive, because those who have done so have had atrophy of the quadriceps muscle, with relaxed knees. There are 12 known failures, recorded in table 5, plus 7 expected disabilities, a total of 19 (12.25 per cent) of 155 cases. Without surgical intervention these men would have been definitely unfit for combat. What the results for these patients will be in five years or after the war, when other factors enter, are yet to be determined. In the meantime, these men have been rehabilitated and man power is conserved. Except for some evidences in cases 30, 80 and 132, there was nothing to indicate beforehand that in these cases results would not be good; age, interval since injury, history and examination are not infallible prognostic points. But if these criteria are favorable, the patient should not be denied the operation. The results of traumatic surgery are not all perfect.

VARIANT DIAGNOSTIC ENTITIES

Hypertrophic Infrapatellar Fat Pads.—This entity is in general only casually mentioned in discussion of derangements of the knee joint. It occurs more often than is realized, and the cause is unknown. The onset is usually gradual, not due to one acute injury. The patient states that the knee has ached or pained for various short intervals or suddenly "given way" while walking. The mechanism of strain and torsion, characteristic of cartilage tears, is generally absent. There is never "locking," although pain may be severe enough to cause guarded motion. Pain is often well localized anteriorly but can be referred with less intensity to the rest of the joint. In the cases of this type encountered in this series, swelling was diffuse, with noticeable increase of joint fluid. A well defined tongue of the hypertrophied pad, of variable consistency up to fairly dense fibrous tissue, is found at operation to extend into the joint space. One can easily see how such a tab can become squeezed between femoral and tibial condyles during extension of the knee, and erosion of the portion of the femoral condyle in contact with this tab is often seen. A mistaken diagnosis of torn meniscus may easily

be made before operation, depending on the history and on the severity of physical signs. Six derangements in this group were so diagnosed, but both the medial and the lateral meniscuses were found intact at operation. The hypertrophic fat tab is easily located, and it is necessary only to bear in mind the possibility of its being present, so that normal cartilages

TABLE 7.—*Return of Function After Correction of Hypertrophic Fat Pad*

	Age, Yr.	Splint, Days	Walk, Days	90 Degree Flexion, Days	Squat on Heels, Days	Duty, Days
Average of 15 cases...	25.40	2.40	6.06	10.06	23.73	31.06

are not removed and so that pathologic conditions may not remain. These cases definitely present more of a problem of convalescence than cases of torn cartilage. Despite a closure of the defect remaining in the infrapatellar fat pad after excision of the hypertrophied portion, there is more reaction of swelling, soreness and synovitis after operation. Functional return is slower, as is shown in table 7.

All the patients of this series who were operated on for correction of hypertrophic fat pads returned to full combat duty, without complaints, except 3, who were reclassified (table 8).

TABLE 8.—*Unsatisfactory Results After Correction of Hypertrophic Fat Pad*

Case No.	Age of Patient	Interval Between Onset and Operation	Pathologic Condition of Knee	Disposition
34	24	8 mo.	Hypertrophic fat pad; chronic synovitis	Reclassified; chronic synovitis
44	23	(?)2 days	Hypertrophic fat pad; joint mouse; osteochondritis of both knees	Reclassified; chronic synovitis
105	21	3 yr.	Hypertrophic fat pad; joint normal	Reclassified; pain; no physical abnormalities

The 3 men who were reclassified are now doing full duty in noncombatant units, including exercises and drill; but 3 reclassifications in 15 cases indicate that 20 per cent of the results were poor. From the surgeon's standpoint, however, there were good results in cases 34 and 105, because the patients after one year on duty have normal knees without physical abnormalities.

Osteochondroma.—Twenty patients with osteochondroma were operated on during that period in which men were inducted for limited service and were never intended for combat duty. The derangements were uniformly long-standing previous injuries with adequate histories and evidence, always verified by roentgenogram. The operations were done because

TABLE 9.—*Restoration of Function in Twenty Cases of Osteochondroma*

	Age, Yr.	Splint, Days	Walk, Days	90 Degree Flexion, Days	Squat on Heels, Days	Duty, Days
Average of 20 cases...	26.55	2.45	6.10	9.40	21.20	26.95

of acute exacerbations, and the patients were most grateful. They have had difficulties for several years, and the annoyance of frequent blocked motion and severe traumatic synovitis was relieved, although when damage to the joint is present one cannot give back a normal joint and cure the soreness caused by eroded joint surfaces. Results in these cases are shown in tables 8 and 9.

Five of 20 patients were given other duty or discharged, indicating 25 per cent poor results. However, these men were not able to do general

TABLE 10.—*Results of Operation for Osteochondroma Leading to Reclassification or Discharge*

Case No.	Age of Patient	Interval Between Injury and Operation	Pathologic Condition of Knee	Disposition
56	23	6 mo.	Joint mouse; osteochondritis dissecans	Reclassified; synovitis and soreness
58	34	2¼ yr.	Joint mouse; osteochondritis dissecans and arthritis	Discharged; arthritis
85	26	7 yr.	Joint mouse; osteochondritis dissecans	Reclassified; synovitis, mild
91	26	8 yr.	2 joint mice; osteochondritis dissecans	Reclassifield; synovitis, mild
100	22	6 yr.	Joint mouse; normal knee	Full duty military police 1 yr.; discharge with recurrent mouse

duty on induction, and for those who were reclassified and still remaining on duty the operations cannot be termed failures. Thus there are but 10 per cent who have not been helped. Fifteen men of this group are now doing full combat duty and have thus been reclaimed from a limited status.

SUMMARY

Operative results in consecutive series of 155 cases of torn semilunar cartilages were analyzed. This analysis showed that lack of immobilization, early exercises and weight bearing give quicker return of function. It indicated that simple excision of bucket handle tears is sufficient, and in so-called complete excision a thin margin of cartilage should remain. One hundred and thirty-two men (85.16 per cent) have returned directly to regular duty on an average of twenty-five days following operation for injuries incurred from a few days to eleven years before. The results justify immediate operation, because the total time lost from recurrence of injury will be considerably more with conservative treatment. Also the longer the torn cartilage remains the more likely it is that joint surfaces will become damaged and that a complete cure cannot be obtained. Hypertrophic fat pads are not generally recognized as a frequent derangement of the knee joint, but operation is warranted because of recurrences of symptoms and possible damage to the joint.